

THE GENERAL PRACTICE MANUALS

- DIAGNOSIS OF ACUTE ABDOMINAL PAIN—*William Requist*
DIAGNOSTIC TESTS IN NEUROLOGY—*Robert Wartenberg*
FLUID BALANCE—*Carl A Mozer*
MANAGEMENT OF THE NEWBORN—*Arthur Hauley Parmelee*
CLINICAL UNIPOLAR ELECTROCARDIOGRAPHY (2D EDITION)—
Bernard S Lippman and Edward Masse
MANUAL OF RHEUMATIC DISEASES—*W Paul Holbrook and Donald F Hill*
MEDICAL MANAGEMENT OF GASTROINTESTINAL DISORDERS—*Garnett Cheney*
ARTERIAL HYPERTENSION (2D EDITION)—*Irving H Page and*
Arthur Curtis Corcoran
CORONARY ARTERY DISEASE—*Ernst P Boas and Norman F Boas*
DIABETES MELLITUS IN GENERAL PRACTICE—*Arthur R Colwell*
ANESTHESIA IN GENERAL PRACTICE (4TH EDITION)—*Stuart C Cullen*
RH ITS RELATION TO CONGENITAL HEMOLYTIC DISEASE AND TO INTRA
GROUP TRANSFUSION REACTIONS—*Edith L Potter*
TREATMENT OF THE PATIENT PAST FIFTY (3D EDITION)—*Ernst P Boas*
OFFICE GYNECOLOGY (6TH EDITION)—*J P Greenhill*
OBSTETRICS IN GENERAL PRACTICE (4TH EDITION)—*J P Greenhill*
DERMATOLOGY ESSENTIALS OF DIAGNOSIS AND TREATMENT—
Marion E Sultzberger and Jack Wolf
PICTORIAL HANDBOOK OF FRACTURE TREATMENT (3D EDITION)—
Eduard L Compere and Sam W Banks assisted by Clinton L Compere
OFFICE TREATMENT OF THE NOSE THROAT AND EAR (3D EDITION)—
A R Hollender
HEADACHE (2D EDITION)—*Louis G Moench*
VASCULAR DISEASES IN GENERAL PRACTICE (2D EDITION)—
Irving Sherwood Wright
OFFICE PSYCHIATRY—*Louis G Moench*
MANUAL OF PROCTOLOGY—*Emil Granet*

(OTHER TITLES IN PREPARATION)

**MANUAL OF
PROCTOLOGY**

MANUAL OF PROCTOLOGY

EMIL GRANET M D

Lecturer Graduate School Columbia University

Visiting Surgeon (Proctology) Sea View Hospital and Associate

Surgeon (Proctology) French Hospital New York Commander (MC) USNR

THE YEAR BOOK PUBLISHERS INC
200 EAST ILLINOIS STREET CHICAGO

Preface

THE PROCTOLOGIST daily sees patients with symptoms pertaining to the anorectal region who have long been subjected to inadequate and even deleterious treatment. Inasmuch as the anorectal region lends itself to simple and accurate methods of physical examination by direct palpation and inspection it is deplorable that many physicians have only rudimentary knowledge of the anorectum, its common lesions and the simple methods for their detection. Experience in graduate teaching has shown that practitioners of medicine are primarily interested in obtaining practical guidance in the examination, diagnosis and treatment of their patients with proctologic difficulties.

With this in mind I have attempted to set forth in simple form the aspects of management of anorectal lesions that are applicable by the non-specialist. I have purposely omitted much of the historical and experimental data readily available in standard text books and have confined myself to comprehensive descriptions of procedures that can be used by the general practitioner or general surgeon with the equipment normally at his disposal. All have been tested in my own experience and inevitably reflect my preferences.

Colon and rectal cancer is dealt with in considerable detail in order to familiarize the reader with contemporary progress in diagnosis, pathologic physiology and the newer operative techniques which enhance the chance for cure by extended excisional surgery. The perplexing problems presented by polypoid disease and ulcerative

	Diet	58
	Laxatives	59
	Hydrotherapy	62
	Electrotherapy	64
5	PEDIATRIC PROCTOLOGY	66
	General Survey	66
	Anorectal Anomalies	72
	Megacolon	75
6	PYOGENIC INFECTIONS OF THE ANORECTUM	82
	Pathogenesis	82
	Cryptitis	88
	Abscess	89
	Anal Fistula	94
7	ANAL FISSURE	103
	Acute Fissure	104
	Chronic Fissure	107
8	HEMORRHOIDS	114
	Etiology and Pathology	114
	Symptoms and Classification	116
	Acute Hemorrhoidal Disease	119
	Chronic Hemorrhoidal Disease	122
9	BENIGN TUMORS	149
	Anus	149
	Rectum Sigmoid Colon	152
10	MALIGNANT TUMORS	172
	Symptoms and Diagnosis	174
	Anal Cancer	180
	Adenocarcinoma of Colon Sigmoid and Rectum	185

	<i>Table of Contents</i>	11
	Lymphosarcoma of Colon and Rectum	208
	Carcinoid Tumors	210
11	ULCERATIVE COLITIS	214
	Chronic Ulcerative Colitis	214
	Circumscribed Hypertrophic Proctitis	229
	Amebic Colitis	230
	Bacillary Dysentery	232
	Antibiotic Colitis	233
12	SPECIFIC INFECTIONS OF THE ANORECTUM	238
	Tuberculosis	238
	Lymphogranuloma Venereum	242
	Gonorrheal Proctitis	246
	Anal Syphilis	247
	Anal Chancroid	249
	Granuloma Inguinale	249
	Anal Warts	250
	Hidradenitis Suppurativa	253
	Actinomycosis	254
13	PRURITUS ANI	257
	Etiology and Pathology	258
	Differential Diagnosis	262
	Treatment	263
	Neurogenic Pruritus Ani	268
14	PROCTALGIAS AND ANORECTAL DYSCRASIAS	272
	Coccygodynia	272
	Proctalgia Fugax	275
	Bizarre Neurogenic Dyscrasias	277
15	PROLAPSE	280
	Mucosal Prolapse	280

Proctology

12	Rectal Prolidentia	281
	Sigmoidorectal Intussusception	284
	Anal Incontinence	289
16	DIVERTICULA OF THE COLON	291
	Symptoms and Diagnosis	293
	Complications	295
	Treatment	297
17	PILONIDAL DISEASE	298
	Etiology	299
	Pathology	302
	Clinical Features	304
	Treatment	318
18	MISCELLANY	318
	Parasitic Diseases of Rectum	322
	Melanosis Coli	323
	Irradiation (Factitial) Proctosigmoiditis	324
	Trauma	327
	Extrarectal Tumors	328
	Fecal Impaction	330
	Foreign Bodies	335
	INDEX	

Anatomy

IN THE EMBRYO the anorectal region is formed from fusion of the ectoderm and endoderm. This fusion results in an extremely complicated anatomic region which still is the subject of intensive anatomic and embryologic studies. In the last decade general uniformity of opinion with regard to the arrangement of fascia, musculature and vascular, lymphatic and nerve supplies has been achieved through the splendid studies of Johnson (1), Milligan and Morgan (2), Levy (3), Gorsch (4) and many others. The description of the essential arrangement of these structures is important because our understanding of pathologic lesions of the anorectum and their management depends on a thorough familiarity with the anorectal structures and their interrelationships. Contemporary surgical procedures which permanently eliminate various lesions with minimum postoperative discomfort and morbidity are based largely on a clear understanding of the anatomy.

Detailed description of individual structures will be omitted as these may be found in all standard textbooks of anatomy. Emphasis will be placed on the relationship of anatomic structures to physiologic function and to the pathogenesis of anorectal disease and its treatment, both medical and surgical.

ANAL CANAL (Fig. 1)

The perianal skin invaginates at the anal verge, thereby forming the anus. The lumen of the anal canal is directed cephalad in

a direction pointing toward the umbilicus. When functioning normally the anus can be distended to a diameter of 3 cm. A cast of the distended anal canal would resemble a truncated cone 2.5 to 4 cm long the anterior surface being shorter than the posterior. At the anal verge the skin gradually changes to become a modified squamous epithelium a transitional tissue conveniently termed the

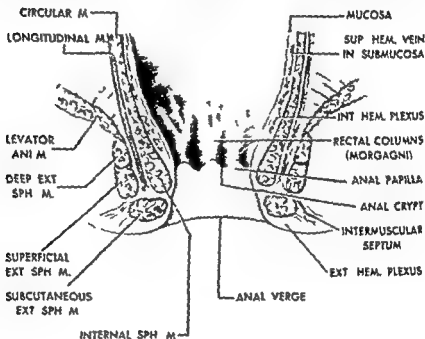


FIG 1 —Sphincteric rectum and anus in coronal section

anoderm. Because of its relative thinness and its close adherence to underlying structures the anoderm is friable and easily traumatized. The anal canal at its proximal limit forms the anorectal line (dentate or pectinate line). The stratified squamous epithelium of the anus at the anorectal line is replaced rather abruptly by high columnar epithelium which forms the mucous lining of the sphincteric rectum. This rectal mucous membrane encompasses a vast number of mucus secreting glands the glands of Lieberkuhn.

The anorectal line is composed of several structures having considerable clinical importance. These include the anal crypts, papillae and valves. Because of the embryologic origin of the anal canal from ectoderm, neoplasms originating in the canal characteristically are squamous cell epitheliomas. In the rectum and colon, malignant neoplasms stem from the glandular mucosa and commonly are adenocarcinomas.

RECTUM (Fig. 2)

The lumen of the rectum is directed posteriorly and follows the hollow of the sacrum. The anterior rectal wall is straight as compared to the rather deep concavity formed by the posterior wall. For practical purposes, the rectum is divided into a lower portion (sphincteric rectum) and a proximal portion (ampullary rectum). The sphincteric rectum extends approximately 4 cm cephalad from the anorectal line; the length of the ampullary rectum varies but rarely exceeds 14 cm. When distended, the ampullary rectum has an average diameter of 6 cm in its midportion. Proximally, the rectal lumen narrows sharply to merge into the sigmoid, this region forming the rectosigmoidal junction. Here the mucosal contour changes from the flat smooth surface of the rectum to the rugated mucosa of the sigmoid. Three semilunar valves (Houston's valves) are present in the rectum, situated on alternate sides of the ampullary portion about 3 cm apart. It is not always easy to visualize the proximal aspect of Houston's valves during proctoscopic examination, so that a polyp or early neoplasm in this location may be overlooked if the examination is not thorough.

The rectum is covered by peritoneum in its superior portion. Anteriorly, the peritoneum is reflected over the bladder and the uterus as well as the rectum, thus forming the rectovesical pouch. In the male, the usual distance of the peritoneal reflection above the anorectal junction measures 7 cm, while in the female the average distance is 4 cm, provided the bladder is empty. The peritoneal folds are reflected diagonally upward and backward to form the leaves of the mesorectum and the mesentery of the sigmoid. The

infraperitoneal portion of the posterior rectum reaches considerably higher than the anterior, being located approximately 14 cm. from the anus. Complete familiarity with the peritoneal reflections as they pertain to the rectum is essential when employing electrotherapeutic procedures in the management of rectal polyps and other

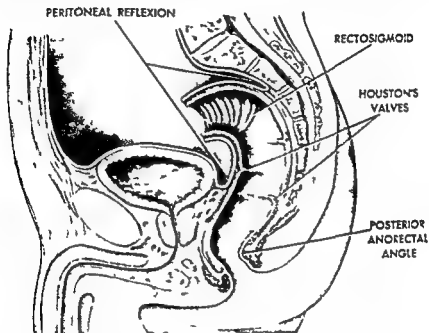


FIG. 2—Distal sigmoid rectum and anus configuration and relationship to peritoneum sacrum and adjacent viscera.

new growths. Above the peritoneal reflection perforations into the peritoneal cavity following deep electrocoagulation of new growths high in the rectum are always possible and such accidents have been reported.

MUSCULATURE

The arrangement of the anal musculature and fascia generally accepted by most proctologists is illustrated diagrammatically in

Figures 1 and 3 In the anal canal and sphincteric rectum proceed ing laterally from the mucosa the veins and arteries of the hemor rhoidal plexus lie in the submucosa The circular muscle of the colon and rectum terminates distally at about the anorectal line as the internal sphincter muscle, a thickened ring of smooth non

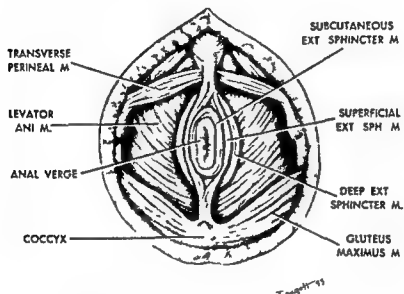


FIG 3—Anorectal musculature seen from below (After Milligan and Morgan.)

striated muscle which completely encircles the anus in its superior portion This muscle is autonomic and is rarely more than 3 mm thick Next laterally the longitudinal muscle of the rectum terminates distally at the anus as the intermuscular septum This musculofibrous band separates the subcutaneous external sphincter muscle from the superficial and deep portions of the external sphincter group Terminal fascial septums of the longitudinal muscle of the rectum also extend laterally through the fibers of the external sphincter muscles serve to support the loose areolar tissue of the

ischioanal space, and terminate by fusing with fibrous elements in the perianal skin

The external sphincter muscles consist of a group of three striated voluntary muscles supplied largely through the somatic sacral plexus. The most proximal the deep external sphincter muscle completely encircles the anorectum at the level of the anorectal line. It merges with the puborectalis portion of the levator ani muscle to form the anorectal ring, a vital element in maintaining anal continence. The superficial portion of the external sphincter lies distal to the deep portion and is contiguous with it only in its lateral aspects. Anteriorly, the fibers of the superficial external sphincter decussate to insert into the perineal body; posteriorly, they decussate to insert into the anococcygeal raphe and then into the borders of the coccyx. This anterior and posterior diversion of the superficial external sphincter creates triangular areas of weakness in the support of the anal canal. This anatomic weakness is critical and is a most important factor in the pathogenesis of anal fissure.

Most caudad of all the subcutaneous external sphincter muscle lies directly under the skin at the anal verge. It completely surrounds the anal canal and is separated from the superficial portion of the external sphincter by the intermuscular septum. The subcutaneous sphincter muscle is of great importance in the surgical management of numerous anal conditions.

The important levator ani muscle (Fig. 4) extends across the deep pelvis as a musculofascial perineal diaphragm. It consists of three overlapping portions: the puborectalis, the pubococcygeus and the iliococcygeus muscles. Anteriorly, both the puborectalis and the pubococcygeus arise from the distal and posterior aspects of the pubic arch and the obturator fascia (white line). They extend posteriorly and medially to encircle and support the posterolateral sphincteric rectum and proximal anus as a powerful muscular sling. Tendinous extensions from the pubococcygeus insert into the ligamentous anococcygeal raphe, thus anchoring the rectum to the perineum. The iliococcygeal portion of the levator arises from the

white line between the obturator foramen and the ischial spine. This relatively thin muscle extends across the deep pelvis behind the rectum, forms the roof of the ischioanal space, and inserts into the sides of the coccyx.

The puborectalis and pubococcygeal muscles function as a contracting sling to pull the sphincter rectum forward toward the

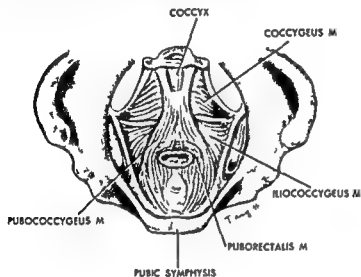


FIG 4—Levator ani muscles and their relationships seen from above

pubis. The posterior anorectal angle is thereby increased and the pelvic aperture consequently narrowed, thus improving sphincter function. Furthermore, the puborectalis in its lateral and posterior aspects fuses with the deep portion of the external sphincter. This composite muscle forms the powerful and important anorectal ring. If this muscle is divided in the excision of high anal fistulas, incontinence will almost invariably result. The rhococcygeus muscle functions largely as a protective diaphragmatic bulwark against the powerful abdominal pressure exerted on the perineum by such acts as coughing, defecation, urination, and parturition.

The levator muscles are important in rectal carcinoma because of the direct spread of malignant cells along the lymphatic and vascular channels that parallel their muscle fibers. Lahey, Jones, Grinnell, and others feel strongly that because of this spread along the levators, abdominoperineal resection for cancer of the rectum is the only procedure which minimizes local recurrence. In this operation of Miles, the levators are widely resected along with the entire rectum and anus.

Spasm of the puborectalis and pubococcygeus portion of the levators is the primary factor in the troublesome syndrome known as coccygodynia. The muscular spasm pulls the coccyx forward with resultant acute flexion of the coccyx, leading to the typical symptoms of coccygodynia: pain on sitting, low back pain, gluteal pain, and pain radiating down the back of the thighs.

VASCULAR SYSTEM

The venous drainage of the anorectal region (Fig. 5) is unique in that it empties into both the portal and the systemic circulation. Vascular drainage is provided for the anorectal region by the important hemorrhoidal plexus. This consists of an abundant and plexiform arrangement of intercommunicating venules extending from the submucosa of the sphincteric rectum through the sup epithelial tissue of the anus to terminate in the subcutaneous areolar tissue of the perianal skin. Although functionally a freely inter communicating venous plexus, the portion above the anorectal line is termed the internal (superior) hemorrhoidal plexus, while the external (inferior) hemorrhoidal plexus comprises that portion of the network below the anorectal line which supplies the anus and perianal tissues.

The inferior hemorrhoidal veins drain the external hemorrhoidal plexus into the systemic circulation via the internal pudendal, hypogastric and iliac veins. The internal hemorrhoidal plexus drains into the systemic and also into the portal circulation. It empties into the small middle hemorrhoidal veins which course through the rectal stalks above the levators to empty into the vena cava via the

iliac veins. The bulk of the drainage from the internal hemorrhoidal plexus takes place through the superior hemorrhoidal veins which empty finally into the portal vein via the inferior mesenterics. This

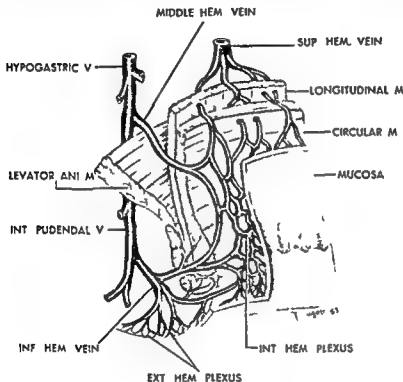


FIG 5—Vascular supply of rectum and anus. veins only are shown. arteries parallel the veins

drainage into the portal circulation explains the common occurrence of early liver metastases from low lying rectal carcinoma. Septicemia characterized by fever and chills commonly results from pyogenic infectious foci in the anorectal region and is ordinarily caused by dissemination of organisms into the systemic circulation.

via the external hemorrhoidal plexus and the inferior hemorrhoidal vein

In man, the almost universal presence of varicosities of the internal hemorrhoidal plexus is attributed to the absence of venous valves in the portal, inferior mesenteric, and superior hemorrhoidal veins. In the upright position, the unsupported weight of a tall column of venous blood exerts great hydrostatic pressure on the internal hemorrhoidal plexus with consequent progressive venous wall dilatation which eventually manifests itself as hemorrhoidal varicosities.

The arterial supply of the anorectal region is distributed mostly to the musculature in this area. The inferior hemorrhoidal artery at its origin from the internal pudendal, is distributed to the ischio-rectal space and the inferior surface of the levator ani. It then divides into terminal branches which supply the external and internal sphincter muscles. The middle hemorrhoidal artery supplies the superior aspect of the levator ani and terminates by anastomosing to a slight extent with the superior hemorrhoidal artery in the sphincteric rectum. The superior hemorrhoidal arteries supply the entire rectum. They terminate as small arteries which descend in the rectal columns of Morgagni to terminate at the anorectal line.

LYMPHATIC SYSTEM

The anatomic distribution of the anorectal lymphatic system (Fig. 6) has clinical significance in the spread of infection and of malignancy originating in this region. Below the anorectal line the subepithelial lymphatic channels consist of a rich, closely woven network which extends well out into the perianal tissues. The lymph is then funneled through collecting vessels which course through the perineum and groin to terminate in the inguinal lymph nodes. Specific lesions affecting the anus and anal verge such as anal epithelioma, tuberculosis, syphilitic chancre, and lymphogranuloma venereum spread directly through this network and distally involve the inguinal nodes. Above the anorectal line and in the ampullary rectum the submucosal lymphatics collect into follicles thence

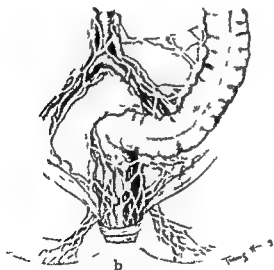
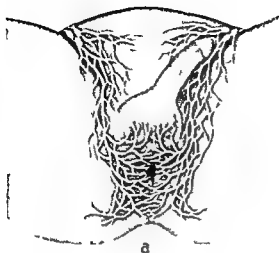


FIG 6—Lymphatic supply of anus rectum and sigmoid *a* anal verge and perianal region drain to inguinal nodes *b* anorectum rectum and sigmoid drain to nodes in lateral pelvis and to periaortic nodes

through collecting vessels reach the extrarectal regional nodes and the nodes in the mesentery. The importance of the extrarectal and mesenteric lymph nodes in the surgical management of rectal cancer is well known.

NERVOUS SYSTEM

The act of defecation depends on complex physiologic interrelationships between the autonomic (sympathetic and parasympathetic) and the somatic (cerebrospinal) nervous systems (Fig 7). Above the anorectal line the rectum is supplied largely by the autonomic nervous system. The sympathetic fibers which supply inhibitory impulses to the wall of the rectum are derived from the lumbosacral sympathetic chain through the hypogastric plexus (pre-sacral nerve). The parasympathetic fibers reach the wall of the

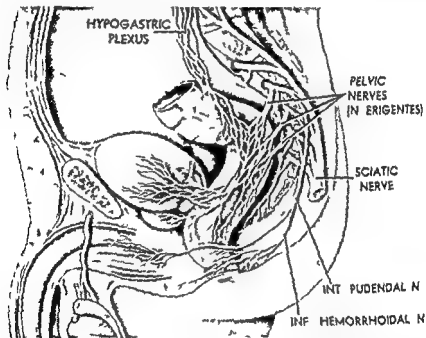


FIG 7—Somatic and autonomic nerve supply of rectum and anus

pathetic) and the somatic (cerebrospinal) nervous systems (Fig 7). Above the anorectal line the rectum is supplied largely by the autonomic nervous system. The sympathetic fibers which supply inhibitory impulses to the wall of the rectum are derived from the lumbosacral sympathetic chain through the hypogastric plexus (pre-sacral nerve). The parasympathetic fibers reach the wall of the

rectum from the sacral plexus (S 2 to S 4) through the nervus erigens (pelvic nerve) and transmit motor impulses. Furthermore, an intramural plexus (Meissner's) present in the submucosa supplies sympathetic impulses to the rectal mucosa. Intramural parasympathetic impulses are carried to the circular and longitudinal muscles of the rectum by Auerbach's plexus. Reuther (5) believes that the termination of the two intramural plexuses in the anus communicates with the somatic terminal fibers of the inferior hemorrhoidal nerves. This would explain the reflex gastrointestinal manifestations commonly seen in painful anal disease.

The somatic (cerebrospinal) nerve supply of the anorectum and the perianal region is derived from the second, third, and fourth sacral nerves through the pudendal nerve. This nerve courses anteriorly and downward through Alcock's canal to reach the ischio-rectal fossa where it gives off several branches as the inferior hemorrhoidal nerves. These are distributed to the anus, the external sphincter muscles, and the immediately adjacent perianal tissues.

Lesions at or below the anorectal line are painful. Those in the rectum are painful when associated with distention or spasm. Therapeutic procedures such as electrodesiccation of new growths and sclerosing submucosal injections which ordinarily are painful can be utilized in the rectum without anesthesia and with little or no discomfort. This anatomic difference in nerve distribution explains why even advanced cancer of the rectum is rarely painful in contrast to the exquisite pain associated with such a morphologically insignificant lesion as anal fissure or a thrombosed external hemorrhoid.

REFERENCES

1. Johnson, F. P. Development of the rectum in the human embryo. *Am. J. Anat.* 16:1, 1914.
2. Milligan, E. T. and Morgan, C. M. Surgical anatomy of the anal canal. *Lancet* 2:1150 and 1213, 1934.
3. Levy, E. Formation of the pelvic viscera. *Am. J. Surg.* 45:357, 1939.
4. Gorsch, R. V. *Perineopelvic Anatomy* (New York: The Telford Co., 1941).
5. Reuther, T. F. Nerves of the anorectal region. *Tr. Am. Proct. Soc.* 41:202, 1940.

Anorectal Symptoms, Examination, and Diagnosis

MOST PATIENTS with rectal difficulties defer medical consultation as long as possible. They do so largely because of fear. They fear examination because of a false sense of modesty induced by a distorted attitude as to what constitutes social refinement. They fear also the physical pain associated with an unskillfully performed rectal examination. Finally many patients delay medical consultation unduly because of fear that their rectal symptoms may be due to cancer. For these reasons, patients consciously or unconsciously refrain from giving information voluntarily regarding anorectal symptoms when a general medical history is being obtained. The possible existence of specific rectal symptoms must therefore be elicited by direct interrogation in the course of obtaining a routine medical history.

A detailed competent and complete examination of the anus and rectum is mandatory when any of three cardinal symptoms are present: (1) recent change in bowel function, (2) defecatory bleeding and (3) anorectal pain. The function of defecation depends upon habitus, nervous temperament, training, diet and other related factors which vary with each individual. For the sthenic, phlegmatic, gourmandizing bartender two or three large soft stools each day are normal. The asthenic, hurried neurotic schoolteacher

may produce a small, hard scybalous stool every second or third day for years and still be in relatively good health. Any persistent change in appearance of the stools or the development of persistent diarrhea or constipation or of both indicates possible organic disease involving the rectum or colon and requires investigation without delay.

Recurrent episodes of bleeding associated with defecation require accurate diagnosis of its origin. Even when hemorrhoids are demonstrated to be the source of bleeding, the upper rectum and the colon must be investigated for other possible sources of bleeding. Too often surgeons see patients with cancer of the rectum or colon in whom the neoplasm had been discovered only after investigation for bleeding which had persisted despite a recent operation elsewhere for bleeding piles. Treatment for hemorrhoids must never be instituted until rectal and sigmoidal neoplasm has been ruled out by careful digital and sigmoidoscopic examination. Too often hemorrhoids and cancer of the terminal bowel coexist.

Anorectal pain is not tolerated with equanimity. Patients with thrombotic external hemorrhoids and anal fissures therefore report for treatment promptly, while patients with rectal cancer will disregard their blood streaked stools and tenesmus lamentably long. Anal and rectal pain requires competent examination to determine its cause. Conditions commonly associated with anal and rectal pain include

- 1 Acute abscesses perianal perirectal ischiorectal supralelevator cryptitis and pilonidal
- 2 Fistulas
- 3 Acute and chronic fissures
- 4 Hemorrhoids acute thrombotic acute thrombotic internal with prolapse
- 5 Specific chronic infection perianal tuberculosis gonorrheal proctitis anal syphilis lymphogranuloma venereum of the rectum granuloma inguinale
- 6 Ulcerative colitis nonspecific dysenteric amebic chronic hypertrophic proctitis

Benign or malignant neoplasms involving the anus, rectum, or pelvis are obvious causes of anorectal pain

Inflammatory and ulcerating lesions of the anus and perianal region are painful because of the somatic nerve supply, rectal lesions are rarely painful in their early stage Extrarectal lesions such as those of the genital tract, can cause anorectal pain In women uterine retroflexion or fibroids pelvic inflammatory disease and genital neoplasms may cause pain which is referred to the rectum In men, prostatic disease commonly is manifested by rectal pain Symptoms such as prolapse anal pruritus perianal elevations, and the presence of pus are obviously of anorectal origin and require thorough investigation

PRINCIPLES OF EXAMINATION

Accurate diagnosis of anorectal lesions can be accomplished by utilizing two principles of physical diagnosis—inspection and palpation A clean and empty lower bowel is necessary for accurate sigmoidoscopy and proctoscopy My experience, both in the hospital and at the office has been that no single method of preparatory cleansing is satisfactory in all cases My office patients take no purges or enemas before examination Digital and anoscopic examination can be performed satisfactorily despite the presence of feces in the rectum If fecal matter is present on anoscopy and is firm and scybalous a glycerin suppository is inserted and sigmoidoscopy delayed until its action has produced evacuation When fecal matter is soft sticky and adherent to the bowel wall it often happens in patients who take mineral oil 120 cc (4 oz) of a stock solution of saturated magnesium sulfate is instilled into the rectum through a funnel and a small rectal tube with the patient in the left Sims position After several minutes the defecation reflex is stimulated resulting in complete evacuation of the contents of the sigmoid and rectum and in a thoroughly clean lower bowel Actual working time is not lost because other patients can be examined or treated in the interim

Since patients requiring proctologic investigation are usually

apprehensive and nervous, examination should be performed with little or no discomfort. The examining tray with the sigmoidoscope and other formidable instruments is best kept covered and so concealed from the patient. As a rule, the examination is started with the patient in the left Sims position thus assuring the patient's comfort and favoring relaxation. A good light, either reflected from a head mirror or a direct spot light, is essential.

INSPECTION

Lesions of the perianal tissues revealed by inspection include the external orifice of fistulas, perianal abscesses, external thrombotic hemorrhoids, external skin tags or tabs, anal fissures, healed scars of former operations, verrucae, condylomas, and the perianal dermatitis associated with pruritus ani. The perianal skin is manually retracted bilaterally with a small gauze pad under each of the examiner's thumbs. The patient is asked to strain down as if you wish to move your bowels. As the patient contracts the abdominal muscles, the external sphincter muscles relax to expose the anal verge. During this maneuver an anal fissure can be clearly seen, a hypertrophied papilla may be extruded, and redundant internal hemorrhoids may prolapse. A great deal of information can be obtained by the maneuver of straining, and its use should never be omitted during a proctologic examination.

DIGITAL EXAMINATION

In catheterizing the male bladder we know that a sphincter spasm is best overcome by allowing the catheter to press gently but firmly against the spastic muscle. Spasm soon relaxes and the bladder can be entered with little pain and no trauma. A similar technique is also employed for the digital examination of the anorectum, especially when sphincter spasm is due to apprehension or to painful anal lesions. The well lubricated, cotted finger is placed firmly at the anal verge and the patient again is asked to press down or strain. With straining by the patient and maintenance of firm digital pressure by the examiner, it will be seen that as the volun-

tary sphincters relax, the anus will thread itself onto the examining finger, thereby enabling the rectum to be entered easily and painlessly

A great deal of information can be obtained by careful and intelligent palpation. The finger is first swung around the anal canal in the region of the anorectal line. Anal stenosis, anal rigidity and sphincter hypertrophy and spasm are readily palpable. Hypertrophied papillae are commonly found. Internal hemorrhoids col-

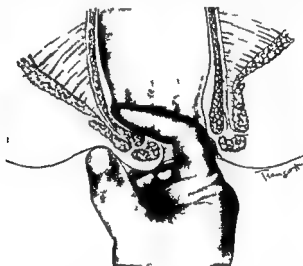


FIG 8 —Bidigital palpation of anorectum

lapse on pressure as do leg varicosities and are usually not palpable except when thrombosed. Polyps, papillomas, submucous tumors or other neoplasms are palpable. Early perianal abscess or cryptitis can be diagnosed by bidigital palpation. In these cases, localized induration and tenderness are found when the perianal tissues are circumferentially palpated by gentle compression between the index finger within the rectum and the thumb or other index finger palpating the perianal tissues (Fig 8). When diagnosed by bidigital palpation, these incipient abscesses can frequently be aborted by conservative treatment.

The ampullary rectum is now examined. This is possible only if the finger can be made to reach it. This can be accomplished by literally jamming the fist well into the soft tissues of the perineum thereby lifting the perineum and consequently increasing the reach of the intraluminal index finger some 5 cm. In attempts to palpate lesions high in the rectum another valuable maneuver consists of

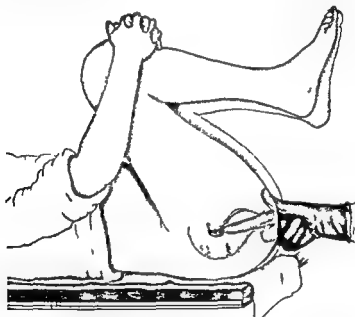


FIG 9 —Palpation of ampullary rectum in flexed supine position

digital examination with the patient lying supine the thighs sharply flexed and pushed down on the abdomen by the patient's fingers interlocked around the knees (Fig 9). This increased abdominal pressure shoves the sigmoid and ampullary rectum deep into the pelvis. With counterpressure on the perineum by the examiner's fist it is possible to palpate lesions as high as 12 cm from the anus.

Extrarectal structures which are palpable through the rectum and which sometimes can be confusing are the cervix, a retroverted

uterus, a prolapsed ovarian tumor, or a prolapsed, pedunculated fibroid, in men hypertrophied prostate and enlarged seminal vesicles may be misleading. Occasionally, a fecal bolus in a redundant sigmoid is palpable through the wall of the ampullary rectum. Sigmoidoscopy or re examination after bowel cleansing will establish the nature of this artefact.

INSTRUMENTS

Endoscopic instruments are required for the visual examination of the anal canal, the rectum, and the distal sigmoid. The anal canal and the sphincteric rectum are best examined by an *anoscope*, a short tubular instrument fitted with an obturator to facilitate insertion. Generally the most useful single instrument is the large size Hirschman type of anoscope (Fig. 10) which has a slanting beveled open end when the obturator is removed. Its large lumen and slanting end visualize almost half of the anorectal lumen at each insertion. Illumination is obtained by the use of a head mirror, or by clip on lights that can be attached directly to the instrument. Insertion of the anoscope, with obturator in place is facilitated by the straining-down maneuver. After the anorectum is viewed the obturator must be re inserted before the instrument is turned to view another quadrant of the anorectum. Turning of the instrument in the anorectum without the obturator in place not only subjects the tissues to trauma from the sharp beveled edges but is painful. Similarly the obturator should be replaced when the instrument is finally withdrawn at the completion of the anoscopic examination. An anoscope of similar construction but with a small lumen is useful in examining patients with fissures or strictures and occasionally in examining infants.

The *proctosigmoidoscope* for general use should have a wide lumen 2.2 cm ($\frac{7}{8}$ in) in diameter. The large bore affords excellent vision for examination and facilitates cleansing as well as intraluminal instrumentation. The standard length of 25 cm (10 in) is adequate. The light should be placed at the proximal end of the sigmoidoscope, the portion nearest the observer's eye. Unavoidable

and frequent soiling of the light with feces blood or pus makes instruments fitted with distal lights impractical. The proximal light when fixed in working position should not unduly obstruct the lumen of the instrument and should be constructed so that it need not be shifted during sponging or other intraluminal maneuvers. The light source is usually house current reduced through a rheostat; if desired the source of electricity can be dry batteries. Finally,

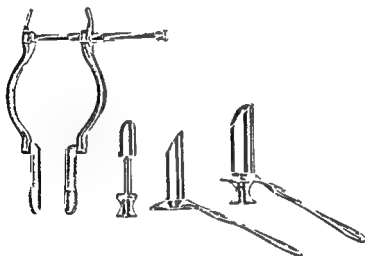


FIG 10—*Left* Newton D. Smith operating anoscope *Right* Hirschman type anoscopes

attachment and removal of the proximal viewing lens and inflating bulb should be easy and rapid to facilitate cleansing and other procedures. In short an ideal sigmoidoscope should be low in cost, have few moving parts and gadgets, a wide lumen, a proximal fixed light, and an easily fitted inflating system. The Yeomans sigmoidoscope (Fig 11) has these features; has withstood the test of time without significant changes in design, and is carried in stock by all surgical instrument supply houses.

An inexpensive spring sponge forceps 30 cm (14 in.) long is

useful for holding cotton ball sponges used in cleaning. Cotton swabs on long applicator sticks are satisfactory in order not to lose the cotton the tip of the stick should be dipped in glue or collodion before the cotton ball is wrapped on. A biopsy forceps is a useful instrument, although not essential equipment for the general prac

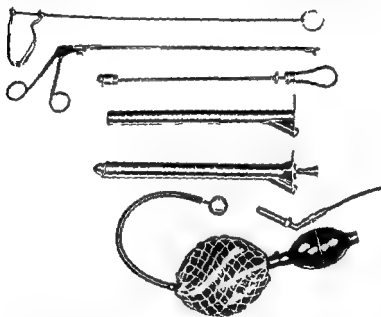


FIG 11 —Spring type sponge holder: Turrell biopsy forceps: Yeomans sigmoidoscopes with inflating bulb

itioner. Turrell's modification of the Yeomans sigmoidoscopic biopsy forceps is a very satisfactory instrument. Endoscopic electrodes and snares for electrocoagulation and electrodesiccation are also useful but should be employed only by those thoroughly trained in their use. These are considered later.

RECTOSIGMOIDAL ENDOSCOPY

Sigmoidoscopy can usually be accomplished with relative ease and with minimal discomfort to the patient. In some individuals

anatomic variations such as marked angulation and redundancy of the distal sigmoid or a sigmoid fixed by a short mesentery make it impossible to pass the rigid endoscope much higher than the recto sigmoidal region. Pathologic lesions such as pelvic inflammatory or neoplastic disease, inflammatory diverticulitis, endometriosis, and bony deformities of the pelvis are among the conditions which limit mobility of the sigmoid, thereby precluding full length sigmoidoscopy. Experienced proctologists are aware of these limitations. Attempts to force the rigid sigmoidoscope past fixed points have in many cases resulted in bowel perforation. Of 94 cases of instrumental perforations collected from the literature by Andresen (1) about half were fatal as a result of this tragic complication of sigmoidoscopy. Actually, an adequate roentgenographic examination by means of a barium enema accurately reveals lesions in the sigmoid and colon above the rectosigmoid junction. Such an examination must be requested for any patient with symptoms suggestive of colorectal disease when visual endoscopic examination of the distal sigmoid is precluded by the factors: mechanical or other, just enumerated.

It must be emphasized that barium contrast enema has little or no value in revealing early disease of the rectum. A negative x ray report in such instances may be dangerous in that it affords the patient a false sense of security. Early neoplastic lesions of the rectum have often been missed because x ray examination instead of sigmoidoscopy was used to determine the cause of rectal bleeding.

The passing of a sigmoidoscope is a maneuver which can be described only in a general way. Facility in sigmoidoscopy depends on experience which is best attained under the tutelage of a competent sigmoidoscopist. After digital and anoscopic examination the patient is placed on the examining table in the knee elbow position with thighs perpendicular to the table top and with the knees separated. For senile cardiac or arthritic patients the left Sims position is utilized. The patient is draped by a sheet folded lengthwise and wound around the buttocks. The lubricated sigmoidoscope with the obturator handle held firmly against the oper-

ator's palm and with the fingers grasping the flange of the scope is pressed firmly against the anus in a line directed toward the umbilicus. The patient is asked to strain down and with firm pressure the instrument is passed through the sphincters into the rectum. The direction of the instrument now is changed to point toward the promontory of the sacrum and the sigmoidoscope is advanced gently until the sacrum limits its advance. It is then withdrawn 3 to 5 cm, thus placing the distal end in the lumen of the ampul

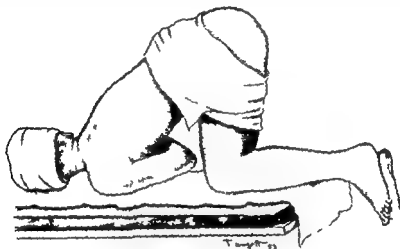


FIG 12—Knee left shoulder position for sigmoidoscopy

lary rectum. The obturator is now withdrawn, the light attached and the focus adjusted. Now by drawing the patient's left arm under his chest by traction on the wrist the patient is shifted to the knee left shoulder position (Fig 12), which further elongates the sigmoid.

The lumen of the rectum is inspected and the instrument is advanced under direct vision to the rectosigmoid (Fig 13). At this point the lumen narrows and the character of the mucosal surface changes from smooth and flat to the corrugated rugae of the sigmoid. Usually the angulation of the bowel above the rectosig

moid makes passage into the sigmoid somewhat painful due to the stretch placed on the bowel and the mesentery. At this point the patient should be warned that he probably will have a transient abdominal cramp. This warning is necessary so that the patient

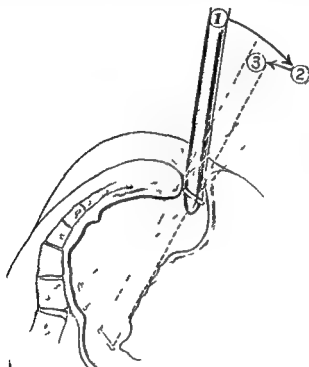


FIG 13—Maneuvers in sigmoidoscopy (Redrawn from Buie)

refrains from a sudden body jerk, the reflex response to pain with its danger of possible instrumental trauma. In fact a running flow of reassuring patter from the examiner during sigmoidoscopy tends to make the patient less tremulous and fearful than otherwise. In sufflation of air into the sigmoid is occasionally necessary to open the contracted or prolapsed intestinal wall, thus re-establishing a visible lumen. In no circumstance should the instrument be advanced

unless an adequate lumen is present Perforation of the lower bowel is particularly apt to occur in senile individuals in patients with ulcerative colitis and in those with lymphogranuloma venereum of the rectum

Detailed examination of the mucosa of the sigmoid and rectum is best accomplished during withdrawal of the instrument The wide lumen of the rectum necessitates movement of the instrument in a circular manner so that all aspects are seen Often overlooked are the proximal aspects of the valves of Houston and the posterior wall of the ampullary rectum Spasm at the rectosigmoid occurs frequently A 2 per cent solution of procaine applied to this area on a large cotton sponge for about a minute often relaxes the obstructing spasm Endoscopic procedures such as bronchoscopy, gastroscopy, or cystoscopy, as well as sigmoidoscopy may be hazardous For this reason the examiner must respect and fully understand the mechanical limitations of the method

REFERENCE

- 1 Andresen A F R Perforations from proctoscopy *Gastroenterology* 9 32 1947

CHAPTER THREE

Anesthesia

THE ANORECTAL region with its relatively localized nerve supply is exceptionally well adapted to the effective use of local or nerve blocking anesthetic technics. This is important for the rich sensory network of this region is responsible for pain, spasm and tenderness out of all proportion to the extent of the lesion. Even minimal inflammatory swellings, fissures and excoriations of the anorectal epithelium are sources of acute discomfort. Patients with painful anorectal lesions should be given the advantage of anesthesia not only during medical or surgical treatment but for manual and instrumental examinations as well.

LOCAL ANESTHESIA OF SHORT DURATION

TOPICAL ANESTHESIA—Many patients delay seeing a doctor for anorectal disease because they dread the pain which from personal experience or from hearsay, they know to be associated with a rectal examination. Unfortunately this is often true. In my opinion to pass an examining finger or instrument into the spastic anus of a patient suffering from an acute anal fissure is brutal. The use of topical anesthesia can eliminate the pain of examination.

Topical anesthesia is effective when applied to acute fissures, erosions and superficial ulcerations of the anal or perianal epithelium. It also is effective when applied to postoperative wounds. To be rapidly effective, the anesthetics must be in aqueous solution.

A useful preparation of this kind is Pontocaine hydrochloride supplied commercially in 2 per cent solution, it has a rapid effect when placed gently in the fissure base on a small pledget of cotton. Crystals of procaine hydrochloride applied to the base of a fissure or ulcer are rapidly liquefied by tissue fluid and their anesthetic effect is immediate and profound. Procaine crystals rubbed into large granulating postoperative wounds, such as those of perianal abscesses or pilonidal disease, facilitate painless removal of exuberant granulation tissue by means of a sharp curet.

ANESTHESIA BY INFILTRATION—Anal pain and sphincter spasm severe enough to preclude rectal examination are often encountered. When inspection reveals no external, open lesion to which a topical anesthetic can be applied, infiltration of a solution of a local anesthetic into the posterior portion of the external sphincter muscles will relax the spasm sufficiently to allow digital and instrumental examination.

Before the injection is started the posterior perianal region is gently palpated to make sure that no subcutaneous inflammatory induration or abscess is present. Infiltration anesthesia is obviously contraindicated if these lesions are found. Following adequate skin cleansing 1 per cent procaine solution in a 10 cc syringe with a 25 gauge hypodermic needle is used to raise a small skin wheal 2 cm from the posterior anal verge. The needle is advanced and 1 cc of the solution is distributed subcutaneously. The hypodermic needle is replaced by a 21 gauge intramuscular needle which is inserted through the wheal and 3 cc of the solution is injected while the needle is advanced a distance of 3 cm toward the midportion of the anus. This places the anesthetic solution in the posterior portion of the external sphincter muscle and should rapidly result in sufficient relaxation to allow gentle introduction into the rectum of the lubricated, cotted left index finger. Then 3 cc of the procaine solution is injected into the sphincter muscles of the right posterior quadrant and the remainder of the solution into the posterior quadrant on the left. During this maneuver the intraluminal finger supports the sphincters and guides the distribution of the solution by

coordinating the direction and depth of the needle thrusts (Fig 14)

Anesthesia by infiltration of procaine solution is effective for minor proctological operative procedures such as excision of thrombosed hemorrhoids, anal papillomas or perianal verrucae. The subcutaneous tissue around the anus is infiltrated completely, then the sphincter muscles in all quadrants are given liberal injections. About

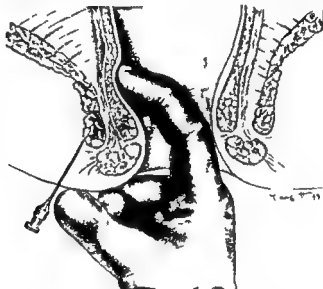


FIG 14 —Technic for infiltrating anal region with anesthetic solution

50 cc of the 1 per cent solution is required and epinephrine is added if not contraindicated medically. Infiltration anesthesia is often necessary to supplement inadequate caudal or spinal anesthesia. Local infiltration anesthesia should not be used for extensive proctologic surgery for the following reasons: (1) it requires multiple punctures in a possibly infected area; (2) it distorts the tissues in the operative field; and most important (3) to be effective a large volume of procaine or other relatively toxic drug must be used.

Merycaine, Nupercaine, Pontocaine, and Xylocaine all produce

a more rapid anesthesia and of longer duration than procaine but they are more expensive and relatively more toxic

REACTIONS—All anesthetic drugs are toxic. Deaths have been caused by cocaine, Butyn, Pontocaine, procaine and some other anesthetics (1, 2). In the case of local anesthetics too rapid injection or careless injection directly into blood vessels may have a toxic effect on the central nervous system due to absorption of high concentrations of the drug into the general circulation.

Mild toxicity occurring early during injection of the anesthetic agent is cerebral in origin. It is manifested by restlessness, apprehension, loquacity, tremors and muscular twitchings, tachycardia and increased frequency of respirations may occur. If the vomiting center is affected, nausea, gagging and vomiting result.

With the onset of any toxic symptoms, injection of the anesthetic drug must be stopped and the patient carefully observed. A rapidly acting barbiturate such as Nembutal sodium, 50 mg per cubic centimeter, given at this stage will usually control progress of the toxicity. It is injected slowly intravenously, 1 cc at a time to a maximum of 4 cc, and its effect on the patient's condition carefully observed. Should toxicity increase and progress to convulsions and to circulatory and respiratory depression, oxygen must be administered promptly to counteract hypoxia. A pressor stimulant such as Vasoxyl hydrochloride is given intravenously in repeated 10 mg doses. The respiratory stimulants (Coramine, picrotoxin) are inactive in combating respiratory depression due to local anesthetic drugs (3).

Reactions due to allergy, hypersensitivity and idiosyncrasy are rapid in onset and caused by even minimal doses of the drug and fortunately are rare. Such reactions are manifested by urticaria, bronchospasm and vasodepression. Symptoms of severe central nervous system toxicity result from minute doses of anesthetic drugs given to hypersensitive individuals.

Toxic and hypersensitivity reactions to local anesthetic agents may be prevented or minimized by adherence to several basic rules of procedure.

1 Obtain a careful history about any unusual experiences with local anesthetics before proceeding with injection

2 Use the drug in the lowest effective concentration and inject it slowly

3 Guard strictly against injection into vascular channels by frequently drawing back on the piston of the syringe during the injection If blood is obtained the direction of the needle should be changed to a position remote from vascular channels

4 When not contraindicated by the presence of hypertension coronary artery disease or hyperthyroidism epinephrine added to the anesthetic solution is of value By its pressor action epinephrine localizes the anesthetic drug and minimizes its systemic absorption Maximal vasoconstriction is obtained with small amounts and minimal dilutions 5 minims of epinephrine in a 1:1000 solution to each 100 cc of procaine solution gives an effective and safe dilution of 1:200,000 (3)

5 Keep the total dose of the drug injected well below the limit of safety for the drug For procaine 1 Gm or 100 cc of the 1 per cent solution is generally considered the dangerous maximal level of safety Such factors as the patient's weight the general status with regard to age and debility and the relative toxicity of the individual drug used must be evaluated in estimating a safe dose

6 Administer a short acting and rapidly effective barbiturate well before the anesthetic solution is injected This will generally minimize apprehension tremors tachycardia and other mild toxic reactions of central nervous system stimulation Nembutal given before procaine administration increased tolerance by about 70 per cent when the procaine was administered subcutaneously or intramuscularly (4) Nembutal 0.1 Gm (1½ gr) should be given orally 1½ hours before operation

7 Careful accurate and constant observation of the patient's general state during and following the injection of anesthetic drugs is mandatory in order to detect the first signs of toxicity and to prepare for their immediate and effective treatment

OF PROLONGED DURATION

OIL SOLUBLE ANESTHETICS—Anorectal surgical procedures generally result in postoperative pain which is truly intense. Post operative inflammatory edema, stimulation of exposed sensory and nerves in the necessarily open anal wounds by chemically irritating rectal discharges and the resulting intermittent sphincter spasms all contribute to the severe anal pain. An anesthetic agent which would safely provide long lasting local analgesia was long sought and was finally successfully introduced as Benacol by Yeomans Gorsch and Mathesheimer in 1927 (5). Many oil soluble anesthetic agents have since been introduced.

A vast and satisfactory clinical experience with oil soluble anesthetics as reported in the literature and by personal communications from many eminent proctologists proves that excellent and prolonged analgesia is attained in proctologic conditions despite the low concentration of most currently marketed preparations (6). The efficacy of the oil soluble anesthetics is unquestionable provided they are carefully used and only when proper indications are present.

Oil soluble drugs are released to the tissues slowly. Proof that anesthetic drugs in oil vehicles are released to the tissues after their injection around an acute anal fissure is clearly provided by the rapid and prolonged analgesia attained in many cases. Although all anesthetic drugs in effective concentration are tissue irritants it has been determined experimentally and clinically that optimal efficiency with minimal tissue reaction is attained by combining procaine base 1.5 per cent ethyl aminobenzoate (benzocaine), 6.5 per cent and benzyl alcohol 5 per cent in pure vegetable oil. Brown *et al* (8) after intensive study, found that sesame and corn oil are superior to peanut and cottonseed oil for intramuscular injection for they are (a) more suitable physically and chemically for this purpose (b) more quickly absorbed from tissue (c) less antigenic and (d) less irritating to tissue.

Examples of oil soluble anesthetics with these qualities are Rectocaine and Anucaine. With both I have obtained satisfactory

analgesia with complete freedom from gross tissue reaction as manifested by induration slough or abscess formation

Because oil soluble anesthetics are tissue irritants the main objection to their use is the very real risk of local tissue destruction slough and abscess formation. However the probability of these complications is remote if the following rules of safety are meticulously observed

1 Select patients for injection carefully. Contraindications are (a) gross active or pyogenic infection in the perianal tissue or in the ischiorectal fossae (b) individuals with poor tissue resistance such as the very senile those with hypoproteinemia and marked avitaminosis and patients with severe heart disease nephritis and diabetes

2 Observe absolute asepsis by thorough preoperative preparation of skin solution syringe and needle

3 Observe the following precautions during injection (a) avoid intracutaneous injection, (b) keep the needle moving as the solution is being slowly injected thereby preventing pooling with its double danger of overconcentration of toxic drugs and increased local tissue tension (c) do not allow the needle to penetrate the rectal or anal canal

4 For postoperative analgesia use the smallest volume of solution which will result in analgesia for no more than about five days. An anesthetic effect prolonged beyond this period is undesirable chiefly because conscious awareness is depressed below the safety level for the control of defecation. Furthermore any anorectal operative procedure which requires analgesic medication after four days (the lag period before active healing by granulation tissue commences) is poorly planned or badly executed

In my own practice the long acting anesthetic agents are used to a limited extent only. They are most valuable in the conservative treatment of acute anal fissure. Following anorectal surgical procedures I use these agents for analgesia in patients with a very high index of pain sensibility. Without analgesia such individuals may need narcotics for several days instead of the usual 24 hours

Proctology

The oil soluble anesthetics are sometimes useful in cases of severe pruritus ani with lacerated perianal skin. The analgesia resulting from the injection will give temporary respite from further compulsive scratching and permit institution of definitive treatment.

NONOLEAGINOUS ANESTHETICS—The disadvantages of the oils as a depository for anesthetic agents have stimulated the search for nonoleaginous anesthetic mixtures with prolonged action.

Diothane hydrochloride—A water soluble complex urethane its use in proctology was popularized by Hertzler (9) and by Roser (10). It is available as 0.5 per cent solution in saline in 5 cc ampules. The technic of administration is that of the oil soluble anesthetics. It gives effective analgesia for about three days.

Efocaine—This preparation was introduced (11) after Monash (12) had demonstrated that water insoluble procaine base deposited in the tissues caused prolonged anesthesia. To make practical use of this principle procaine base and butyl aminobenzoate were dissolved in propylene glycol with just enough water added to form a saturated solution. When this solution is injected tissue fluids provide enough water to precipitate the water insoluble anesthetic drug at the site of injection thereby forming a repository depot.

The indications for the use of Efocaine in proctologic conditions the technic of injection and the precautions to be observed are the same as for the oil soluble anesthetics. A number of favorable reports have appeared (13-15) while others (16) note the occasional occurrence after its use of sloughing and abscesses around the anorectum. My impression based on the related experiences of many proctologic surgeons who have used Efocaine is that this product has no decided advantages over the time tried oil soluble anesthetics.

CAUDAL EPIDURAL BLOCK

The extradural injection of an anesthetic solution through the sacral hiatus a convenient and highly satisfactory method of block.

ing the sacral nerves is ideal for proctologic surgery. It affords complete relaxation of the musculature and excellent anesthesia and its extent is limited to the saddle area of the perineum. Caudal anesthesia unlike spinal anesthesia is not accompanied by post lumbar puncture headache. This complication is common in proctologic cases probably because of very early ambulation its frequency varying from 6 per cent (17) to 30 per cent (18). The possibility of neurologic complications following spinal anesthesia though infrequent must also be kept in mind (19).

The disadvantages of caudal block are (1) the relatively prolonged time before onset of effective anesthesia (2) the relatively large amount of drug required (3) the danger of inadvertent intravenous administration of the drug or of unintentional intraspinal administration (4) occasional failure of anesthesia because sacral anomalies prevent retention of anesthetic solutions within the sacral canal (20).

TECHNIC (Fig. 15) —The patient is placed in the prone jack knife position. After careful skin antisepsis the caudal canal is entered with a spinal needle in the usual manner (21). Precautionary aspiration to insure against intravenous or intraspinal injection is followed by the injection of 5 cc of 1 per cent Metycaine with added epinephrine. After a three minute pause in order to make certain that the needle point is not in the spinal canal the patient is asked to raise his heels toward the ceiling. The ability to do this is positive proof that the injection is extradural. In the inverted position aspiration of the spinal fluid is not a reliable test.

During this necessary wait the second sacral nerves are blocked with 10 cc of Metycaine on each side. This is done by introducing a spinal needle through an anesthetized skin wheal 1 cm medial and 2 cm distal to the superior posterior spine of the ilium directing the needle cephalad medially and ventrally at a 45 degree angle to the skin surface. It will enter the second sacral hiatus and should be advanced about 3 cm before start of the slow injection of the solution while the needle is gradually withdrawn.

At the end of the three minutes 30 cc of the solution is slowly

remaining immobile in the prone position is obvious, that it is also practical is demonstrated by our experience with caudal block. Spinal saddle block can be performed in the inverted position by making use of hypobaric solutions of anesthetic agents (22 23)

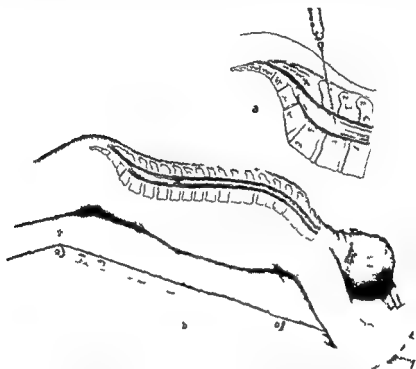


FIG 16—Spinal saddle block *a* 2 mg of hypobaric Nupercaine hydrochloride solution administered with patient in operating position. *b* solution floats upward and localizes around sacral nerve roots in cauda equina

With the cooperation of the Department of Anesthesiology at the French Hospital New York City, we have instituted a technic for spinal saddle block anesthesia with a hypobaric Nupercaine hydrochloride solution (24) This method has the following advantages

- 1 Administration of anesthesia by thecal puncture with the

patient already in the preferential position for proctologic surgery—the prone jackknife position

2 Accurate localization of the anesthetic around the sacral nerve roots in the *cruda equina* (Fig 16)

3 Minimal amount of toxic drug utilized 2 to 3 mg of Nupercaine ■ compared to at least 50 mg of procaine

4 At least 90 minutes of anesthesia

5 Low incidence of postlumbar puncture headaches Because the patient is maintained in the inverted position spinal fluid pressure ■ negative or very low leakage of the fluid through the puncture point ■ negligible and early sealing of the puncture wound ■ favored

We use Nupercaine hydrochloride in a 1:1500 solution 0.66 mg per cubic centimeter (the hypobaric solution of Howard Jones) This solution available in ampules has a specific gravity of 1.0036 at 37 C. which is hypobaric in spinal fluid (sp gr 1.005)

The patient is placed prone on the table at a 10 degree jackknifed position After adequate skin preparation a point 1 cm medial and 1 cm cephalad to the posterior superior spine is anesthetized A 22 gauge spinal needle is inserted and directed cephalad medially and ventrally at about a 45 degree angle to the plane of the skin surface The needle point slides over the lamina at the base of the fifth lumbar spine to penetrate the dura in its posterior lateral quadrant at the level of the fourth lumbar vertebra (Figs 17 and 18) If spinal fluid does not flow spontaneously the obturator is replaced the upper half of the table is elevated to a slight Fowler position and the patient is asked to raise his head This maneuver increases the pressure of the spinal fluid and when the obturator is again removed a free flow of spinal fluid should result The table ■ again cranked down to ■ 10 degree Trendelenburg position and 3 cc of Nupercaine solution is slowly and continuously injected over a 10 second interval The patient ■ kept in this position throughout the operation Satisfactory saddle anesthesia of the perineum is attained in two to five minutes

against the face by the patient himself. With the face mask properly fitted the patient takes about 30 breaths. By this time a sufficient quantity of the volatile drug has been inhaled to afford ample analgesia for minor, rapid procedures, such as incision of an abscess or removal of packing (25)

REFERENCES

- 1 Shumacker H B. Reactions to local anesthetic agents. *Surgery* 10 119 1941
- 2 Mayer E. Fatalities from local anesthetics. *JAMA* 90 1290 1928
- 3 Sadove M S *et al*. Reactions to local anesthetic agents. *JAMA* 148 17 1952
- 4 Cripp L H and de Castilio Riberio C. Allergy to procaine hydrochloride with three fatalities. *JAMA* 151 1885 1953
- 5 Yeomans F C, Gorsch R. V and Mathesheimer J L. Benacol in pruritus ani. *M Rec* 127 19 1928
- 6 Smith T E. Relief of pain following anorectal surgery. *South M J* 36 630 1943
- 7 Emery F F, Mathews C. S and Schwabe E L. Absorption of siltbestrol and theelin from sesame and peanut oils. *J Lab & Clin Med* 27 622 1942
- 8 Brown W E, Wilder W N and Schwartz P. A study of oils used for intramuscular injections. *J Lab & Clin Med* 29 259 1944
- 9 Herrvler A E. The use of diothane in the control of afterpain in hemorrhoidectomy. *Am J Obst & Gynec* 27 301 1934
- 10 Rosser C. Diothane in surgery of the anal canal. *Surg Gynec & Obst* 59 820 1934
- 11 Ansbro F P *et al*. Development of Efocaine: a new approach to prolonged local anesthesia. *Anesthesiology* 13 306 1952
- 12 Monash S. Prolonged local anesthesia by injections with procaine base. *J Invest Dermat* 14 79 1950
- 13 Raicus E. Postoperative anesthetics in anorectal surgery. *M Times* 80 347 1952
- 14 Tucker C. C. Control of postoperative pain in anorectal surgery. *J Kansas M Soc* 52 230 1952
- 15 Gross J M and Shaftel H E. Role of Efocaine in anorectal anesthesia and analgesia. *New York State J Med* 52 1413 1952
- 16 Personal communications from F Wyman, M M Marks, I Bernstein, S Schapiro and R Turell
- 17 Carmel A G. Spinal anesthesia in proctology. *Am J Surg* 79 144 1950
- 18 Jennings W A and Karabin, J E. Incidence of headache following spinal anesthesia. *Am J Surg* 46 317 1939
- 19 Kennedy F *et al*. Grave spinal cord paralysis caused by spinal anesthesia. *Surg Gynec & Obst* 91 585 1950
- 20 Trotter M and Letterman G B. Variations in the sacrum: their significance in caudal anesthesia. *Surg Gynec & Obst* 78 419 531 1944

- 21 Lundy J S A method for producing block anesthesia of the sacral nerves
Am J Surg 1 262 1928
- 22 Dye F C and Vaughan J A Saddle block anesthesia hypobaric for proctological surgery Anesthesiology 10 189 1918
- 23 Gómez Alonso E Técnica de la puncion lumbar en decubito prono con soluciones hipobaras de pantocaina al 0.5% Arch Soc estud clin. Habana 41 309 1951
- 24 Granet E and DePolo E J Hypobaric sacral anesthesia in anorectal surgery New York State J Med 53 2211 1953
- 25 Hewer C L Trichloroethylene as an inhalation anesthetic and analgesic Canad M A J 62 324 1950

CHAPTER FOUR

General Therapy

DAILY, the medical practitioner is deluged by literature extolling the virtues of one product or another. Add to this the reports of clinical studies with these new products and therapeutic confusion is the result.

MEDICAMENTS

Therapeutic drugs in suppositories, ointments, and in solution are commonly applied to lesions involving the sphincteric rectum, the anus, and the perianal region. Such lesions include anorectal proctitis, cryptitis, hemorrhoids with erosion, fissures, and pruritus ani. The essential purpose of most of the suppositories and ointments is to supply analgesia, antiseptics, and, possibly, to stimulate epithelization. Their sedative action results from the effect of a topical anesthetic drug, usually benzocaine, on exposed end nerves or on mucosa. Their use on an external thrombotic hemorrhoid covered by intact, edematous skin is obviously ineffectual and irrational.

SUPPOSITORIES—A venerable and popular vehicle for the rectal administration of drugs, the suppository, is often improperly used for lesions in and adjacent to the anus. But as a result of pain or burning, reflex sphincter spasm holds the anal canal tightly closed, and little if any medication contained in a suppository remains at the site of the disturbing lesion during or after insertion of the suppository into the rectum. Proctoscopy performed an hour

or more after insertion of a suppository reveals the melted cacao butter (oil of theobromin) to be smeared over the rectal ampulla.

Use of the opium and belladonna suppository, although some surgeons still employ it after operations on the anus is obsolete. These narcotic and anticholinergic drugs have no local sedative action and are better absorbed when administered orally. The suppository is useful as a vehicle for administering sedation and other medication to patients with nausea and vomiting or with motion sickness and to infants and children. Nembutal as well as aminophylline suppositories are examples. Before insertion a suppository should be slightly melted under warm tap water.

OINTMENTS—For proctologic use these are dispensed commercially in collapsible tubes equipped with a screw-on pile pipe. These preparations contain a topical anesthetic agent in a vehicle of lanolin and petrolatum or a hydrophilic water soluble base. Antiseptic drugs may be incorporated.

For mild analgesia Nupercainal, Eucupin, Diothane and Pontocaine ointments are satisfactory products. Wyandoid ointment, Stymex cream (water washable), Tricainal ointment (with Pyri-benzamine) and Benadex ointment or cream are good products for analgesia with antipruritic effect. For analgesia and antiseptics, Surfactaine and Penicillin G ointment and Trynazin ointment (with sulfathiazole, water washable) are useful. For topical treatment of lesions in the ampullary rectum, Rectalgan (an analgesic in oil) may be used. Since the perianal skin of certain patients is or may become hypersensitive to one or another of the drugs in these preparations, atopic skin reactions must be carefully watched for when these ointments are being used. A pile pipe is useful for distributing the ointment within the anal canal and the sphincteric rectum.

Ambulatory patients with postoperative anal wounds are generally made more comfortable if the wound is coated with a soothing ointment. Carbolated petrolatum is satisfactory and inexpensive. Cod liver oil or chlorophyll in ointment bases supposedly stimulate the healing of sluggish wounds (1) but the evidence of this

beneficial effect in anal lesions is not convincing. The disagreeable odor of the fish oil and the green staining by the chlorophyll are definite disadvantages of these ointments.

Exuberant granulation tissue occurs in open wounds which heal by granulation such as those following saucerization of perianal abscesses or of pilonidal disease. Granulation tissue proliferates less profusely in clean wounds. When wounds are dressed with an ointment containing urea and the sulfonamides granulation tissue is held in check. It has been demonstrated that urea renders sulfonamide compounds more soluble; it also has a solvent action on pus, debris and necrotic tissue which act as sulfonamide inhibitors (2). A useful product containing sulfonamides and urea peroxide is Triple Sulfa Cream.

An agent useful for its lytic effect on thick purulent exudates, such as occur in large perirectal and pilonidal abscesses, is Varidase which contains the lytic enzymes streptokinase and streptodornase. It is used in solution as a topical agent for enzymic and phagocytic debridement of purulent material (3).

DIET

The relative comfort of a patient who has painful anorectal lesions is directly dependent on the physical and chemical characteristics of the stool mass passed and the frequency of defecation. This applies also to patients with recent postoperative wounds. To a large extent the nature of the fecal mass depends on the nature of the food ingested. If food intake is insufficient scybalous pellets formed in the distal end of the colon tend to pack in the rectum with consequent pain and trauma by their eventual forced passage through the anus. When the diet contains a preponderance of the cellulose legumes such as cabbage, Brussels sprouts, corn, celery and the salad greens the stools tend to be bulky and hard. Large amounts of milk produce a large, dry, crumbly stool.

Patients with painful anal lesions often try to limit the size of the stool by avoiding solid foods. Many take large amounts of fruit juices. The subsequent laxation of the bowels produces frequent and

repeated excoriating liquid dejections. The high trypsin content of these alkaline diarrhetic evacuations excoriates and digests the exposed anal lesions thereby retarding healing. Patients with ulcerative anorectal disease and those with postoperative wounds should be fed an adequate diet high in protein, fat and vitamins but low in roughage. Tablets of ascorbic acid 100 mg daily will provide the vitamin C so necessary for proper wound healing. Irritating spices and alcohol in quantity are barred. Fat in the diet is digested down to irritating organic soaps which act as mild laxatives. A diet of this type theoretically should result in a daily stool of good size soft though formed and chemically bland.

The secretion from the mucosa of the sphincteric rectum in most patients with pruritus ani and perianal dermatitis has a pH of 8 to 10. It is probable that this highly alkaline secretion is a significant irritating factor which favors the persistent excoriation of the perianal skin. I have been able repeatedly to reduce the alkaline reaction of the rectal mucosa to pH 5.5-6 by dietary therapy. This is done simply by implanting lactic acid-producing bacilli in the colon through the addition to the daily diet of dairy products containing large amounts of *Bacillus acidophilus*. Such products include buttermilk, *Acidophilus* milk, sour cream, Yogurt and lactose. Trilactic (polymolecular lactic acid with lactose) given four times daily in tablespoon doses aids the growth of *B. acidophilus*. This dietary regimen must be maintained for a prolonged period because the lactic acid-producing bacilli are easily supplanted by other intestinal organisms if not constantly nurtured. A lowered pH of the rectal secretion may be a very important factor in attaining remission in stubborn cases of pruritus ani.

LAXATIVES

Despite an adequate bland low residue diet satisfactory defecation does not occur daily in most patients with painful anal lesions or postoperatively. Laxation becomes necessary and is often a problem because its proper regulation is achieved with difficulty.

Many patients with anorectal disease daily ingest a considerable

quantity of liquid petrolatum in an attempt to obtain a soft lubricated stool. Mineral oil is undoubtedly an effective lubricant, but when it is used as an emollient for a long time it becomes a hazard to health. The deleterious effects of mineral oil from a proctologic point of view were first emphasized by Morgan (4) and Thiele (5), and later by Becker (6). The following objectionable effects are important:

1. Mineral oil suspensions of pathogenic bacteria may be forced into the anal crypts of Morgagni during defecation. The stage is thereby set for an ensuing cryptitis or abscess.

2. Fat soluble vitamins are absorbed from ingested food and excreted with the oil, and a deficiency of vitamins A, D, E and K may result. With a low absorption level of vitamin K the prothrombin level of the blood is depressed. Abnormal bleeding from hemorrhoids in mineral oil addicts may be due in part to this deficiency.

3. The normally empty rectum becomes a constant reservoir for an emulsion of oily feces. Consequently the important structures at the anorectal line are constantly bathed in pathogenic material. Fresh postoperative wounds are likewise continually contaminated and healing is thereby probably delayed. Leakage through the anus of oily infected feces is common, and is a significant factor in setting up a contact chemical dermatitis in the perianal skin, an important etiologic factor in pruritus ani.

4. Absorption of food is ineffectual because the action of the digestive enzymes is interfered with by the oil coating on the food particles. A considerable amount of mineral oil is absorbed into the liver during this process; the oil clogs the lacteals in the bowel wall and many lacteals are rendered incapable of performing their proper function—the absorption of food fats.

Mineral oil should therefore be used for only short periods as an emollient. Administered as a retention enema in quantities up to 240 cc (8 oz), liquid petrolatum is often useful postoperatively to lubricate the lower sigmoid and rectum and possibly to soften desic-

cated stools Use of olive oil for this purpose seems needlessly extravagant

The patient with a postoperative wound finds defecation less uncomfortable when the stool mass is formed but soft and glairy and can be passed intact as a single bolus The hydrophilic colloids and the methylcelluloses both administered orally with a liberal fluid intake and a regular but bland diet produce a stool mass having satisfactory physical properties The hydrophilic colloids commercially available include psyllium karaya and the alginates The mucilaginous element in these preparations distends 30 to 40 times its volume in water and is responsible for the lubricant action Cass and Wolf (7) in a critical clinical evaluation of laxatives found the psyllium preparations most satisfactory methylcellulose in tablet form was generally unsatisfactory My own experience parallels theirs

The hydrophilic laxatives are available commercially as Konsyl Mucilose (concentrated) and Siblin all are psyllium products Metamucil and Hydrocil contain up to 50 per cent lactose or dextrose the function of which is to aid dispersion in the liquid vehicle The addition of sugar actually halves the yield of pure colloid per standard dose and so doubles the cost to the patient of each effective dose Kalpin is one of the alginate hydrophils The daily dose of these preparations varies from 1 to 3 heaping teaspoonfuls taken in a large volume of water The dose must be regulated to suit the needs of the individual patient When the hydrophils alone fail to produce a stool of satisfactory consistency 1 or 2 teaspoonfuls of a mild saline laxative such as Phospho Soda (Fleet) taken daily will increase the fluid content of the colon and permit increased hygroscopic absorption by the colloids

Prune juice taken in considerable quantity has become a popular and effective laxative Its action is due to the relatively large content of isatin an irritating cathartic present in most commercial preparations of prune juice Its habitual use as with all irritating laxative drugs results in spastic hyperirritability of the colon

HYDROTHERAPY

Of the simple therapeutic measures for relieving anorectal pain the time honored sitz bath is generally the most satisfactory. In patients with postoperative wounds it relieves sphincter spasm, stimulates local vascularity, and cleans the wound surfaces of purulent exudate and fecal discharges. The sitz bath is universally used following proctologic surgery. It is also used to diminish discomfort due to painful anorectal lesions such as cryptitis, thrombotic or strangulated hemorrhoids, and anal fissuration. The sitz bath can be taken several times daily, in a bathtub or even in a dishpan. It should be comfortably hot with maximal temperature 48 C. (110 F.) the depth of the water should be no higher than 15 cm (6 in.) and immersion time should not exceed 10 to 15 minutes. Hotter, deeper, and longer sitz baths are weakening and may cause syncope. A valid objection to the postoperative sitz bath as it is usually taken, in a bathtub, is the unavoidable contamination of the immersed portion of the body by the filthy, purulent exudates washed from the postoperative anal wounds. The further spread of contamination to bath towels is obviously unsanitary, and the frequent changes of linen strain laundry facilities.

Hydrotherapy in the form of the perianal douche (Fig. 19) has many advantages over the sitz bath and none of its disadvantages. A running stream of warm water cleanses the wounds and stimulates local circulation more thoroughly, it is more convenient in that it saves linen and for the ambulatory patient saves time. The douche is self administered by the patient who sits over the edge of the tub and hoses himself down with warm tap water delivered at the anus by means of an inexpensive rubber hose bath spray available in any pharmacy. The wide spray tip is removed so that the water emerges in a solid stream directed at the anus by the patient who holds the hose in his right hand while retracting the left buttock with the left hand. The position and the function of the hands are interchanged frequently. About three minutes for each session is sufficient.

Rigid perianal hygiene is generally advised as an important phase in the management of patients with pruritus ani. It consists merely of cleansing the perianal skin with absorbent cotton and warm water following defecation. The chemically irritating feces are thus washed from the raw and fissured perianal skin. If toilet tissue is used the fecal matter is actually inoculated into the sensitive perianal skin. In cryptitis and fissure pruritus ani and other inflammatory lesions affecting the anal canal the presence of soft

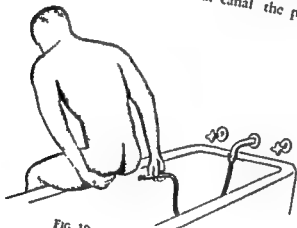


FIG 19—Technic of anal douche

fecal matter remaining constantly in the sphincteric rectum or anus following defecation is deleterious and obviously undesirable. Rectal and anal hygiene instituted by means of postdefecatory rectal lavage affords comfort to the patient and cleans the residual fecal matter from the anorectal lesions. Rectal lavage is administered by injecting warm tap water into the rectum by means of a 2 oz infant's rubber enema syringe and immediately expelling this. Residual fecal matter contained in the distal rectum and anus is thus washed out. The lavage is repeated two or three times until the returned fluid as seen in the toilet bowl contains no fecal particles. Hot moist packs of saline solution or of dilute witch hazel solutions applied to fresh postoperative wounds are very soothing. Flan-

nel pads saturated with a solution of witch hazel and glycerin are commercially available as Tucks Nursing care may be minimized by using a pillow to prop an electric pad or hot water bag against the moist dressings. Wet dressings kept cold with an ice cap are useful to reduce edema and are very comforting in cases of thrombotic or prolapsed hemorrhoids. Burow's solution, 1:40, makes a very satisfactory wet dressing for the treatment of inflammatory perianal dermatitis resulting from severe pruritus and/or the dermatitis which often occurs after the ingestion of the broad spectrum antibiotics.

ELECTROTHERAPY

In contemporary proctology this modality is employed only in the treatment of rectal and lower sigmoidal polyps. These lesions are effectively destroyed by electrodesiccation or electrocoagulation applied with special electrodes through an endoscope (see Chapter 9).

Electrosurgery in some form formerly enjoyed a questionable vogue. For such procedures as hemorrhoidectomy, surgical diathermy using coagulation or desiccation was lauded as a means for performing bloodless surgery while sterilizing the operative site. However, delay in separation of the necrotic stump caused inordinate edema and pain, frequent late hemorrhage, and prolonged healing time. For similar reasons cauterization of anal fissures and ulcers is also obsolete. The cautery knife is used by some proctologists in the office for excisional surgery of anorectal lesions such as hemorrhoids, fissures and fistulas. The patient is sent home directly after operation, fortified against pain by one of the long acting injectable anesthetic agents. Generally, this form of ambulatory surgery is inadvisable, because the patient is deprived of the constant and competent postoperative care and observation so necessary in the critical early postoperative days.

The electrotherapeutic management of certain types of anal strictures by galvanism has been revived by Feit (8). The galvanic current, a direct or continuous electrical flow with positive or nega-

the polarity possesses true chemical action. The water of the tissue fluids is hydrolyzed so that the negative oxygen radical collects at the positive pole while the hydrogen element travels to the negative pole. The negative active electrode is an olive shaped copper ball which is placed and held against the stricture during the 12 minute treatment. A solvent effect on the scar tissue is presumed to take place which enables easy dilatation. Treatments are repeated at intervals of about six days. Feit reported satisfactory results in 24 cases. The evaluation of this modality for the treatment of anal strictures must await confirmatory favorable reports.

Medical diathermy by short or microwaves applied to the anorectum is helpful in conditions which require relaxation of spastic musculature and conditions associated with inadequate vascularity. (9) In the treatment of anal sphincter spasm and also in coccygodynia caused by spasm of the levator constrictor muscles sedative diathermy is valuable following digital massage. Diathermy properly applied does increase the arterial blood flow through the tissues. Applied to indolent pilonidal or fistulectomy wounds improved vascularity through diathermy may help epithelization.

REFERENCES

- 1 Turell R. Cod liver oil ointment therapy in proctologic disorders. New York State J Med. 50 2282 1950
- 2 Holder H G and Mackay E M Wound therapy with reference to carbamide sulfanamide mixtures in contaminated wounds. Mil Surgeon 90 509 1942
- 3 Miller J M *et al* Clinical experience with streptokinase and streptodornase. J.A.M.A 145 620 1951
- 4 Morgan J W Misgivings on mineral oil as a laxative. Am J Surg 42 360 1938
- 5 Thiele G H. Mineral oil and saline laxatives. South M J 35 920 1942
- 6 Becker G L. The case against mineral oil. Am J Digest Dis 19 344 1952
- 7 Case L J and Wolf L P. Clinical evaluation of certain bulk and irritating laxatives. Gastroenterology 20 149 1952
- 8 Feit H L. Treatment of anal stenosis by galvanotherapy. J Internat Coll. Surgeons 18 98 1952
- 9 Krusen F H. Medical applications of microwave diathermy. Proc. Royal Soc. Med 43 641 1950

Pediatric Proctology

CONSIDERABLE PROGRESS has been accomplished in recent years in the study and in the management of proctologic conditions in children. The discussion here is limited mainly to those conditions in which recent studies have clarified our concept of their pathologic physiology, management or treatment.

GENERAL SURVEY

Proctologists examine relatively few children during any one year. This is why too often proctologic lesions are found in children only after examination to determine the cause of repeated rectal bleeding, mucosal prolapse and severe constipation. Congenital deformities are soon noted, often first by the parents. A valuable contribution to our general knowledge of pediatric proctology was made by Schapiro (1), who reviewed all pediatric admissions to a general hospital over a 35 year period. He found that 2,700 (or 2.34 per cent) of the total pediatric hospital admissions were for proctologic lesions. Of these, 1,457 were for diarrheal diseases and 139 for congenital anorectal anomalies exclusive of spinal and sacral deformities. In the latter group, 50 infants or children had an imperforate or absent anus in some form. Fissure occurred in 129 children, abscess in 35, fistulas in 20. Polyps were found in 101, rectal or sigmoidal prolapse in 83. The cause of rectal bleeding was undetermined after complete investigation in 31 children.

In a report on the proctologic examination of 127 consecutive private pediatric patients common lesions found by Mentzer (2) included fissure in 31 prolapse in 11 abscess and fistula in 10 and polyp in 6 In 100 consecutive children examined by Murray (3)

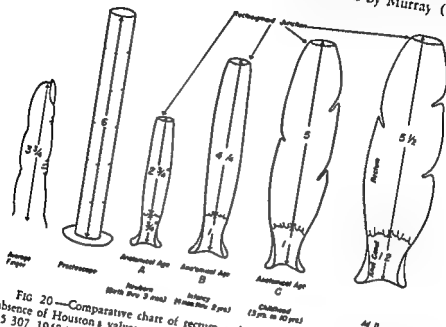


FIG 20—Comparative chart of rectum and anal canal during growth note absence of Houston's valves in infancy (From S Schapito Rev Gastroenterol. 15 307 1918)

constipation and fissuration were found in 32 prolapse in 18 abscess and fistula in 8 and polyp in 6

I have cited these reports in detail to emphasize the fact that infants and children should be examined specifically for proctologic lesions when symptoms appear Except for hemorrhoids trouble some proctologic lesions like fissure abscess and polyps are not unusual in children Prolapse of the sigmoid into the ampullary rectum and even external rectal prolapse is common in infants and children

Visual and digital examination is readily performed with the infant or child firmly held in the Sims position. With the lubricated cotted index finger bearing gently, steadily, and firmly against the anal verge, the sphincter soon relaxes completely to allow adequate digital examination of the rectum and sigmoid (Fig. 20). For endoscopy, the child is inverted on a jackknifed table, the parent firmly holding down the chest and arms while the office assistant holds the legs and thighs. Anesthesia in infants is not desirable or necessary, for the examination is not ordinarily painful and should be accomplished purposefully and rapidly. A sigmoidoscope of $\frac{3}{8}$ or even $\frac{7}{8}$ in diameter easily passes a digitally dilated anus in an infant. In older children, a 0.12 Gm (2 gr) Nembutal suppository inserted an hour before examination allays apprehension. The infant sigmoidoscope with a $\frac{1}{4}$ in lumen is superfluous equipment for routine examination. Its field of vision is so limited that rapid accurate examination of the entire rectum and sigmoid is difficult. Anoscopic examination can be performed easily with instruments of adult size unless congenital stricture is present.

Problems of elimination, the diarrheas and constipation occurring in infants and children are usually the concern of the pediatrician. The findings on endoscopy in the diarrheas of children differ little from those found in adults, and for this reason will not be separately considered.

RECTAL BLEEDING—In infants and children some form of bleeding associated with defecation is the usual symptom which indicates the need for proctologic examination.

FISSURE—When bleeding is intermittent and associated with painful passage of a hard, overlarge stool in constipated infants and children retraction of the buttocks and visual inspection will usually reveal an anal fissure or ulcer. Bleeding and pain are suffered when the lesion is torn open by the passage of the fecal bolus. The pain causes sphincter spasm which blocks defecation, hence, rectal constipation and eventually the large desiccated traumatizing stool. As in adults examination of the rectum is deferred until the adjacent portion of the sphincter muscle is anesthetized with procaine.

If endoscopy reveals no other pathologic lesions the sphincter muscle is blocked by an analgesic with prolonged action as is done for an acute fissure in the adult (Chapter 7). An anticonstipation regimen is instituted, and if necessary a regular daily 2 oz. plain warm water enema is administered to induce defecation. By so minimizing further anal trauma the fissure usually heals spontaneously. Should the fissure persist further conservative therapy is futile and definitive surgery becomes mandatory.

POLYPS.—The parent occasionally notes fresh blood smeared on or mixed in the fecal bolus. Defecation is not painful and constipation need not be present. In such cases sigmoidoscopy sometimes reveals a single polyp usually pedunculated in the distal sigmoid or rectum. Polyps may however be multiple. These are obvious causes for rectal bleeding. Just as in adults the individual polyps are excised surgically if low and accessible they are excised by cautery snare through the sigmoidoscope if high in the rectum in the sigmoid. If the polyps are sessile they are destroyed by electrocoagulation. Multiple polyposis is familial hereditary and usually is noted in older children. Its management constitutes a serious problem the various aspects of which are considered in detail in Chapter 9.

PROLAPSE.—Bloody mucus partly coating the stools or passed as spontaneous dejections is a common symptom in infants and preschool children. These children have a colicky diathesis some are constipated and defecation requires inordinate straining. The mucus originates in hypersecreting cells of an edematous granular, injected redundant mucosa. Repeated trauma caused by intermittent prolapse of the sigmoidal mucosa through the narrowed rectosigmoid eventually results in mucosal erosion and capillary bleeding. In external rectal prolapse erosion results from the constant trauma of prolapse through the anus.

The frequency of rectal prolapse in infants is well known (4). It is also known that this condition does not persist but gradually disappears as the child attains his third year. Prolapse in infants is due in part to developmental factors which include relaxation of rectal

ligaments as well as looseness of the posterior rectosacral fascia.

In infants and young children the ventral aspect of the sacrum is virtually flat and the coccyx has little forward tilt (Fig 21, *b*). In the infant, the rectum is a straight tube, with very little of the lateral and posteroanterior curve seen in the adult. Consequently a line drawn from the superior strait of the pelvis would virtually drop out of the anus because the long axis of the rectum parallels

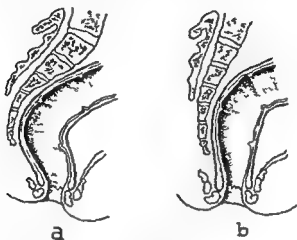


FIG 21 —Sacral curve *a* posterior rectal wall supported by deep sacral curve in older children and adults *b* underdeveloped curve in infants and young children favoring rectal prolapse

the flat sacrum and coccyx. This lack of support by surrounding tissues apparently explains the ease with which defecatory straining favors an unsupported downward excursion of the terminal bowel and in some infants eventual extrusion of the bowel through the anal orifice (5).

The commonest form of prolapse occurs in the rectal ampulla and does not protrude through the anus. It is palpated easily as a circular sulcus formed by invaginated thickened intestinal wall. The intussusceptum of the prolapse is seen sigmoidoscopically as a cervix like structure of edematous injected granular and friable sigmoidal mucosa.

Although rectal prolapse is seen in very young children (below the age of 2 years) this condition disappears as a clinical entity as the immediate preschool age is reached. It again makes its appearance in adults as simple external mucosal prolapse and less commonly as procidentia a prolapse of all coats of the intestine. There is an obvious and logical explanation for the disappearance of prolapsus in older children. After the second year the sacral curve deepens and as the levator muscles develop they pull the coccyx forward. By this mechanism the important posterior anorectal angle described by Bure is developed and concomitantly the axis of the anal canal is angled posteriorly (6). The major force of defecation therefore is now expended on the posterior rectal wall supported by a deep sacral curve (Fig 21 a) and no longer on the sphincteric rectum as was the case in infancy. With this shift of expulsive force directed increasingly away from the anus the rectal mucosa no longer prolapses. Internal or sigmoidorectal prolapse tends to persist and though at times asymptomatic can at all times be demonstrated (7).

Even though external prolapse generally subsides spontaneously with growth it is a troublesome condition which causes considerable anxiety to the parents. Active therapy is best managed by the pediatrician. It is directed toward regulating the function of defecation so that excessive expulsive force is avoided. Diarrhea must be controlled and constipation dispelled. The hydrophil colloid laxatives made into an emulsion with milk and added to cereal feedings have adequately solved the constipation problem in many children. At the onset of treatment regular strain free defecation should be instituted by instilling a warm tap water enema with a 2 oz hand bulb syringe usually about 10 minutes after the first morning feeding. When external prolapse occurs it should be replaced immediately by the mother or nurse. With a folded wash cloth wrung out in hot water used as a pad firm steady pressure is exerted on the externalized bowel this usually readily replaces the prolapsus. One ounce of warm mineral oil gently instilled as a retention enema nightly is helpful. The parents should be constantly reassured as to

the transient nature of the condition and its spontaneous subsidence with the child's growth I have not found it necessary to resort to more radical treatment such as submucosal sclerosing injections or linear cauterization of the mucosa by actual cautery. An excellent description of these measures has been given by Bacon (8).

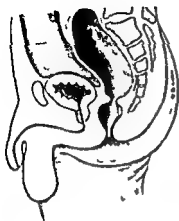
ABSCESS AND FISTULA—In view of the prevalence of the diarrheal disorders in infants it is remarkable that these conditions are not more common, particularly because the preformed intramuscular glands, ducts and anal crypts generally considered the source of most pyogenic perianal abscesses, are well developed at birth. Of 1,500 fistulas surgically treated by Mitchener (9), only 12 occurred in children below the age of 13. These lesions, when they occur in children, should be treated as in adults. Abscesses should be drained immediately, then saucerized. Fistulas should be excised.

ANORECTAL ANOMALIES

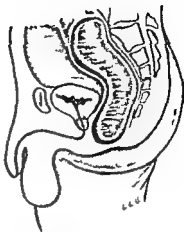
It is impossible to estimate accurately the incidence of anorectal malformations. The collected statistics by Schapiro (10) show an incidence of 1 in 1,650 based on almost 700,000 pediatric hospital admissions.

I have had little personal experience with the serious and extensive problems presented by these conditions. The interested reader is referred to the authoritative work of Ladd and Gross (11), whose classification of the anorectal anomalies has been most generally followed. The work of Santulli (12) and his associates at Babies Hospital, New York, on the management of these difficult conditions demonstrates the value of present-day surgical methods in the radical and well planned procedures which have increased the yield of cures and lowered mortality.

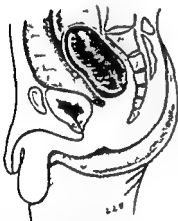
Figures 22 and 23 clearly illustrate the Ladd and Gross classification of anorectal anomalies. The incidence of each type according to Schapiro's 1,154 collected cases, is: type I 11.6 per cent, type II 21.6 per cent, type III 63.6 per cent, type IV 3 per cent. Fistulas occur most often in type III lesions. Of these, the rectovaginal fistulas were about four times as frequent as the rectoperineal, rectourethral, and rectovesical fistulas.



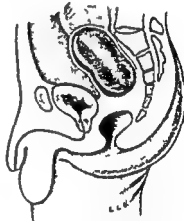
Type I



Type II



Type III



Type IV

FIG 22 —Malformations of anus and rectum Type I stenosis at anus or lower rectum due to incomplete rupture of anal membrane Type II membranous form of imperforate anus due to persistent anal membrane Type III anus imperforate and rectum ending as blind pouch a varying distance from perineum Type IV caudal and lower rectum forming pouch separated for a varying distance from blind rectal pouch (Figs 22 and 23 from T V Santulli Surg Gynec & Obst. 95 601 1952)

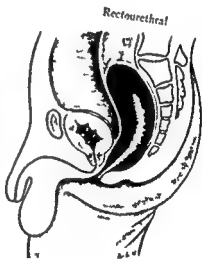
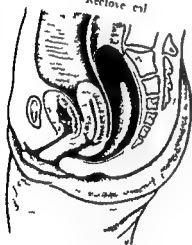
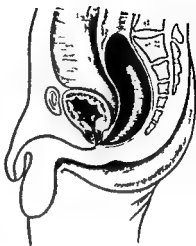
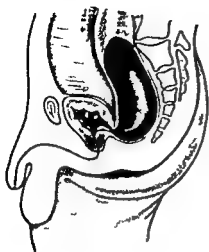


FIG 23 —Fistulas in Type III imperforate anus

Associated extrarectal anomalies are common they were present in 28 per cent of Bacon and Sherman's (13) series and in 34 per cent of Santulli's 62 cases. Major co-existing anomalies such as congenital heart disease, esophageal atresia and tracheoesophageal fistula may be directly responsible for postoperative mortalities. In cases of imperforate anus the method of outlining the rectal pouch by roentgenograms taken with the newborn in an upside-down position as advocated by Wangensteen and Rice (14) is widely used. Performed after approximately 18 hours of age the colonic gas is presumed to rise in the rectal pouch and the distance from the metal marked anal dimple can be estimated. Meconium in the distal end of the rectal pouch may result in an erroneous estimate of the distance of the rectum from the anal dimple. When fistulas are present the gas may escape from the rectum thereby making diagnostic aid by roentgenography impossible.

The treatment of anal stricture by dilatation with graduated dilators of rubber followed by finger dilations is usually satisfactory. It should be routine practice for all obstetricians and pediatricians to pass a finger into the rectum of the newborn infant so that if stricture is present treatment can be begun immediately. For operative procedures and other phases of management of congenital anorectal anomalies the reader is referred to the comprehensive studies of Ladd and Gross (11), Santulli (12) and Bacon (13).

MEGACOLON

The pathogenesis of this important but relatively uncommon condition has been clarified only recently so that the literature is filled with conflicting theories with regard to its causes and management. On the assumption that the colonic dilatation resulted from an autonomic imbalance lumbar sympathectomy was performed in a number of cases but the clinical improvement was most often haphazard and not definite enough to make the operation an accepted procedure. Medical treatment with the anticholinergic drugs like atropine and Mecholyl similarly offered no consistent therapeutic result.

In 1948, Orvar Swenson of the Children's Hospital Boston basing his concept of Hirschsprungs disease on the fundamental neuropathologic studies of Tiffin *et al*, of Whitehouse and Kernohan and of others developed a surgical treatment for Hirschsprungs disease which for the first time resulted in consistent cures Bodian *et al* in England Ehrenpreis in Stockholm Hiatt Lee *et al*,

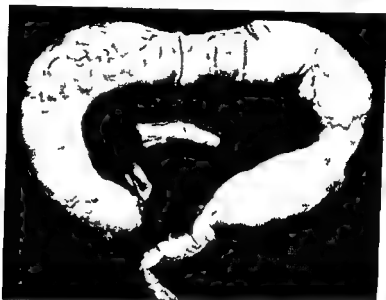


FIG 24 —Hirschsprungs disease colon and rectum with segment of normal colon in center for comparison (Figs 24 and 25 from F H Whitehouse and J W Kernohan Arch Int Med 82 75 1948)

State and others in this country have fully confirmed Swenson's work. In 140 collected cases the operation was successful in 133 failed in three and four patients died (15). These important contributions to our knowledge of colon affections are so fundamental that they warrant further discussion.

Megacolon in children occurs in two distinct clinical and pathologic forms. Hirschsprungs disease and idiopathic megacolon. Although dilatation of the descending and proximal colon is present in both forms dilatation extends down to the anus in idiopathic

megacolon whereas in Hirschsprung's disease the rectum and distal sigmoid are narrowed (Fig 24). They are easily differentiated by roentgenographic contrast enema, whereby the narrowed distal segment of Hirschsprung's megacolon can be consistently demonstrated. Hirschsprung originally described the disease in 1887 and stated that the large intestine appeared somewhat dilated, only the rectum was not enlarged but rather seemed to be the site of some kind of narrowing. The attention of most workers in this disease has been focused on the dilated segment and this erroneous approach was responsible for much subsequent confusion in the description of the pathogenesis and treatment of Hirschsprung's disease. Ehrenpreis (16) in 1916 showed that the dilatation is not congenital but develops weeks or months after birth. He considered megacolon to be a result of dysfunction in evacuation really an achalasia of the distal segments. An example of a similar mechanism is the well known idiopathic cardiospasm. Fawcett (17) pointed out that the megacolon in infants which follows obstructing lesions in the rectum is more marked than the colonic dilatation in adults resulting from chronic obstructive lesions in the rectum such as those caused by lymphogranuloma venereum. He showed experimentally that maintained constriction of the sigmoid in young puppies caused far greater dilatation of the bowel than similar experimental constricting lesions in adult dogs. It appears therefore that the dilated colon of children's megacolon is a result of chronic obstruction in growing infants.

Tiffin and associates (18), and later Whitehouse and Kernohan (19) studying autopsy material established the fact that all normal elements of the myenteric plexuses of Auerbach and Meissner are present in the dilated portion of the colon while in the narrowed distal segment (sigmoid and rectum) ganglion cells are completely lacking in both Auerbach's and Meissner's plexuses (Fig 25). The latter finding has been confirmed by Bodian and co-workers (20) by histologic examination of 28 cases. Swenson and associates (21) studied colonic motility in patients with Hirschsprung's disease. They recorded strong peristaltic waves in the dilated and hyper

trophied colon but the narrowed distal segment showed only increased tonus and peristalsis was absent. This is clear evidence that normal myenteric ganglion cells are lacking in the distal segment.

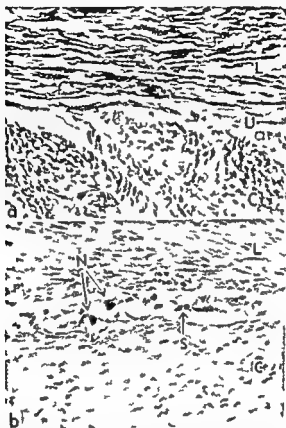


FIG 25 —Hirschsprung's disease *a* section from rectum shown in Figure 24 note absence of myenteric plexus of rectum *L* longitudinal muscle *U* usual site of plexus *C* circular muscle *b* ganglion in transverse colon shown in Figure 74 *N* nerve cell *S* supporting cell

From these physiologic and pathologic studies Swenson (22) concluded that removal of the pathologic achalasic distal segment should be the logical and practical treatment for the disease. This he proceeded to carry out as a one stage rectosigmoidectomy with

suture of the descending colon to the sphincteric rectum just above the anorectal line. When this operation is performed on children with true Hirschsprung's (achilasic) megacolon cure is attained and postoperative x rays show restoration of colonic size and tonus to normal. State (23) has modified Swenson's operation by excising the dilated portion of the colon then restoring continuity by an intraperitoneal suture of the transverse colon to the ampullary rectum.

Pathologically, roentgenographically and clinically idiopathic megacolon differs from Hirschsprung's disease. The etiology of idiopathic megacolon is unknown but it is presumed to result from faulty bowel habits, anal sphincter spasm, autonomic imbalance with overactivity of the sympathetics and perhaps endocrine dyscrasias. Roentgenographically the bowel dilatation starts immediately above the anus and includes the rectum and sigmoid. No abnormalities have been demonstrated in the myenteric plexuses. Clinically the dilatation develops slowly; morbidity and mortality are far less than in cases of Hirschsprung's megacolon and the nutrition is well maintained. Attacks of acute obstruction are rare whereas in Hirschsprung's disease they are frequent and may be the cause of death.

Idiopathic megacolon may respond favorably to the spinal anesthetic test. The hyperactive sympathetic system is inhibited by the induction of spinal anesthesia. The unaffected parasympathetic system then induces expulsive contractional efforts in the bowel which may result in copious evacuations. This positive demonstration of autonomic imbalance with overactivity of the sympathetic system indicates that a lumbar sympathectomy would be helpful and possibly curative. Other measures useful in treatment include bilateral partial sphincterectomy of the internal sphincter muscle as reported by Jenkins (24), sphincter dilatation, colonic irrigations, intelligent laxation and administration of 0.1-0.2 Gm (1 1/2-3 gr) of Mecholyl bromide (a parasympathetic stimulator) once or twice daily. If dilatation is extreme, segmental resection of a part of the colon may be necessary.

REFERENCES

- 1 Schapiro S Occurrence of proctologic disorders in infancy and childhood *Gastroenterology* 15 653 1950
- 2 Mentzer C G Pediatric proctology *South M J* 41 798 1948
- 3 Murray F H Discussion on Mentzer (2)
- 4 Schapiro S Applied anatomy of infants and children in proctology *Rev Gastroenterol* 15 307 1948
- 5 Daniels E A Rectal disorders of childhood *Am J Dis Child* 54 573 1937
- 6 Buie L A *Practical Proctology* (Philadelphia W B Saunders Company 1938) p 111
- 7 Goldman C The normal excursion of the sigmoid into the rectum *Tr Am. Proct Soc* 34 85 1933
- 8 Bacon H E *Anus Rectum Sigmoid Colon* (3rd ed Philadelphia J B Lippincott Company 1949) p 508
- 9 Mitchener P H Some notes on fistula in ano especially in children *Brit. J Surg* 2 364 1914
- 10 Schapiro S The incidence and regional classification of the congenital anomalies of the ano rectum (Mimeographed 1951)
- 10 Schapiro S Scientific Exhibit A M A Atlantic City 1951
- 11 Ladd W E and Gross R E *Abdominal Surgery in Infancy and Childhood* (Philadelphia W B Saunders Company 1941)
- 12 Santulli T V The treatment of imperforate anus and associated fistulas *Surg Gynec & Obst* 95 601 1952
- 13 Bacon H E and Sherman L F Surgical management of congenital malformations of anus and rectum *A M A Arch Surg* 64 331 1952
- 14 Wangenstein O H and Rice C O Imperforate anus *Ann Surg* 92 77 1930
- 15 Ehrenpreis T Rectosigmoidectomy for Hirschsprung's disease *Acta chirurg scandinav* 102 251 1951
- 16 Ehrenpreis T Megacolon in the new born *Acta chirurg scandinav Supp* 112 1946
- 17 Fawcett B Relation of the factor of growth to the pathogenesis of megacolon megalum and megaduodenum *Surgery* 29 491 1951
- 18 Tiffin M E Chandler L R and Faber H K Localized absence of ganglion of myenteric plexus in congenital megacolon *Am J Dis Child* 59 107 1940
- 19 Whitehouse F R and Kernoohan J W Myenteric plexus in congenital megacolon *Arch Int Med* 82 75 1948
- 20 Bodian M Stephens F D and Ward B C H Hirschsprung's disease *Lancet* 1 19 1950
- 21 Swenson O Rhineland H R and Diamond I Hirschsprung's disease A new concept of the etiology *New England J Med* 241 531 1949
- 22 Swenson O A new surgical treatment for Hirschsprung's disease *Surgery* 28 371 1950

23. Sate H Surgical treatment for idiopathic congenital megacolon (Hirschsprung's disease) Surg Gynec & Obst 95 701 1952
24. Jenkins J A Hirschsprung's disease Australian & New Zealand J Surg 17 189 1948

Pyogenic Infections of the Anorectum

PYOGENIC INFLAMMATORY disease involving the anorectum consists of a triad which usually commences in an anal crypt and spreads through the duct system to involve the tissues adjacent to the anorectum to form an abscess which when drained of its pus contracts to terminate as an anal fistula

PATHOGENESIS

The anal crypts (Fig 26 *a*) their tributary ducts glands and the adjacent papillae located as they are at the level where anal dilatation occurs are subject to both trauma and infection. Despite the protection afforded by overlapping redundant folds of rectal mucosa anal valves lymph follicles and local tissue immunity active inflammation does occur (Fig 26 *b*). Infection of the anal crypts is often associated with the habitual use of mineral oil and saline laxatives. Thiele (1) pointed out that in these individuals during defecation in the phase of anal dilatation an oily or liquid suspension of fecal bacteria is forced into the anal crypts and possibly into their tributary ducts. Acute cryptitis when it occurs is dependent on the virulence and number of the implanted organisms, local trauma and other factors pertaining to lowered resistance to infection. With inflammatory changes in the crypt and its tributaries the adjacent papilla becomes edematous and indurated. The presence of hypertrophied anal papillae is presumptive evidence that there is chronic infection in the underlying anal

crypts although no visible sign of active inflammatory cryptitis is present

Originating in the deep apex of an anal crypt an epithelium lined tubule or duct proceeds laterally through the subepithelial tissues penetrates the internal sphincter muscle and terminates in the sphincteric connective tissue as a multilocular intramuscular



FIG 26—Anal crypt and duct. *a* normal *b* in subacute cryptitis (From C. L. Kratzer *Am J Surg* 79:34, 1950)

gland. There are four to eight duct-glandular systems in the human anus. After penetrating the internal sphincter muscle these glands spread out below the inferior surface of the levator ani, thus affording access of infection through the ischioanal space. Occasionally the ducts penetrate the levator ani to terminate in intramuscular glands which lie above the levator. Bacterial infection from the anus can thus be channeled to the supralelevator space to result occasionally in the formation of a supralelevator abscess. Evidence exists that an infection can take place in the gland even though

there is no demonstrable connection with the anus by means of the anal ducts (2 3) For this reason, during operation for fistula it is sometimes impossible despite diligent and careful search with probe or by injection of tracer dyes to find the internal orifice of a fistulous tract

Perianal abscess usually results from infection caused by pyogenic staphylococci streptococci and colon bacilli in the lower bowel which invade the perianal tissues by means of the anal duct-

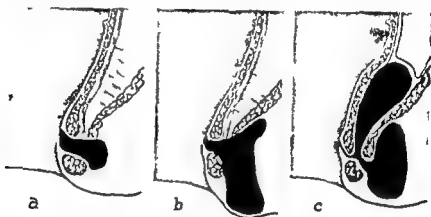


FIG 27—Anorectal abscesses *a* ischioanal *b* ischiorectal *c* supralevator and ischiorectal

glandular system (4) These abscesses are classified by their anatomic location as submucous, intramuscular, ischiorectal and supralevator (Fig 27)

Approximately 90 per cent of perianal abscesses stem from pyogenic crypt infections The remaining 10 per cent comprise (1) abscesses secondary to anal fissures (2) abscesses of hematogenous metastatic origin, (3) those following anal trauma from impalement or from swallowed foreign bodies and (4) abscesses which occur as complications of specific intestinal infections such as tuberculosis ulcerative colitis regional ileitis and lymphogranuloma venereum of the rectum These are discussed later in the respective chapters dealing with these conditions

When an acute perianal abscess has been drained of its pus either by incision through the perianal skin or by spontaneous rupture the resulting wound contracts to form a sinus tract. This

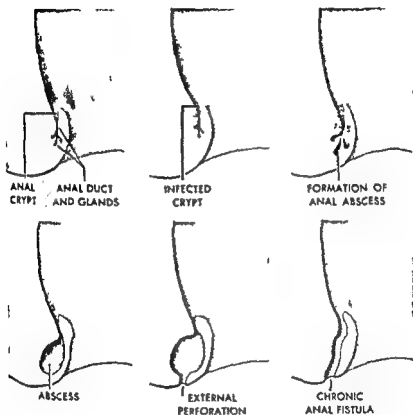


FIG 28—Pathogenesis of anal cryptitis, abscess and fistula (Redrawn from Neisefrod)

end result of a drained abscess is known as an anal fistula. The anatomic course of the fistula follows the ramifications of the pre-existing abscess. Thus a simple fistula with a single tract usually results from incision or spontaneous drainage of a localized anal or ischio-rectal abscess. This type of simple direct fistula usually has

its internal orifice in an anal crypt and the external orifice on the perianal skin. The sequence of events in the formation of a simple anal fistula has been well demonstrated graphically by Nesselrod (Fig 28)

Complicated fistulas are common and are exemplified by the



FIG 29—Chronic perianal abscess with multiple (watering pot) fistulas

horseshoe fistula. This results from an ischiorectal abscess which extended through the posterior communicating space under the puborectalis muscle to involve the opposite ischiorectal fossa. Occasionally the granulation tissue at the external orifice of a chronic fistula will epithelize and close. As a result of this barrier external

drainage ceases. The original abscess which burned out long before and is now of low virulence slowly recurs. When it again penetrates the integument it may do so by reopening the external fistulous orifice. Often however the abscess bursts its original confines in one or more tracts and after devious ramifications penetrates the skin at one or more points somewhat distant from the

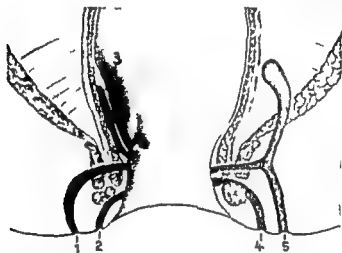


FIG 30—Anatomic relationships of usual types of fistulas: 1 high level anal 2 subcutaneous 3 submucous 4 low level anal 5 supralelevator and ischiorectal (Redrawn from Gabriel)

original fistulous opening. Repeated recurrences of this process result in a chronic perianal abscess with widespread multiple watering pot fistulous orifices (Fig 29). Similarly reforming abscesses may burst through the levator muscles and fascia to extend into the pelvirectal or retrorectal supralelevator spaces. These complicated fistulas pose therapeutic problems which often require staged operations. The most comprehensive classification of anorectal fistulas (Fig 30) is probably that of Milligan (5). Infected granulation tissue is frequently found between contiguous fistulas.

CRYPTITIS

A localized point of tenderness palpable by digital examination in the anal canal is suggestive of acute cryptitis. When visualized through an anoscope an acutely inflamed crypt is characterized by turgescence and redness of the adjacent epithelium. Occasionally a small drop of mucus can be expressed. Exploration by hooked probe during examination increases trauma and serves no useful purpose. Symptoms are usually present only during the acute phase of pyogenic cryptitis. Patients complain of a sense of burning and aching in the anus which is worse following defecation. Sphincter tenesmus frequently is present. As the acute inflammation in the crypt subsides so also do the symptoms. Spontaneous resolution when it occurs, requires approximately a week.

Chronically infected anal crypts are clinically important as a source of focal infection in systemic diseases. Their importance has been held to be comparable to infection in the teeth, tonsils, and prostate. (6) Cryptitis and papillitis have been considered the inciting cause of reflex disturbances of gastrointestinal functions. These reflexes, relayed to the autonomic nervous system through the synapsing fibers of Meissner's and Auerbach's plexus in the anus, can conceivably cause changes in both peristaltic and secretory functions of the colon resulting in cramps, constipation, flatulence and excess secretion of mucus.

To my mind, the enthusiasm with which some authors implicate one or more diseased crypts as the *bête noire* responsible for remote gastrointestinal symptoms, arthritic and muscular pain in the lower back and pelvis, fatigue and other symptoms suggestive of neurasthenia is rarely justified.

Acute cryptitis is treated medically. Sphincter spasm is allayed by hot sitz baths or frequent warm perianal douches. The trauma of defecation is avoided by instilling into the rectum a small enema of warm tap water just before defecation and repeating it once or twice directly after bowel action to cleanse the sphincteric rectum and anus of irritating residual fecal matter. Mineral oil should be

avoided but hydrophilic colloid laxatives may be used. An anesthetic, antiseptic ointment deposited in the anal canal by means of a pile pipe several times daily helps allay spasm and promotes resolution. Suppositories are locally ineffectual in conditions affecting the anal canal because little, if any, of the drug remains in the anus. Sitz baths or warm anal douches are taken several times daily if possible. These measures relax sphincter spasm and reduce venous stasis. Penicillin 600 000 units and streptomycin 1 Gm, daily for several days may prevent abscess formation and should hasten resolution.

Subacute and chronic cryptitis are usually associated with other such benign anal lesions as hemorrhoids, fissure, proctitis and pruritus ani. Infected anal crypts should always be excised concurrently with other anal lesions at operation.

ABSCESS

SYMPTOMS AND DIAGNOSIS—The cardinal symptom of perianal abscess is pain. The location of the abscess determines the intensity of the pain. A small abscess involving the sphincteric region is characterized by severe pain because spasm of the musculature exerts constant tension on inflamed tissues in a highly sensitive region. An abscess in the loose areolar tissue of the ischioanal space which contains few somatic sensory end nerves, can attain considerable size before severe discomfort is manifested. Similarly in a supralevator abscess pain is usually a late symptom.

Fever, prostration and even chills may be early symptoms of perianal or perirectal suppuration. These probably result from pyogenic emboli originating in the abscess and distributed to the portal or systemic venous systems by the hemorrhoidal plexus.

Early lesions are manifested on palpation by an area of firm, tender induration localized to the immediate vicinity of the anal canal and the sphincteric muscles. An inflammatory exudate resulting from the presence of tissue edema, bacteria, lymphocytes, and leukocytes is present but little necrosis has as yet occurred. Usually one sees the patient when the abscess is perceivable as a large, firm,

tender indurated mass most often situated in the ischio-rectal fossa. Redness local heat and fluctuation are later physical manifestations of the inflammatory process. These indicate that central necrosis has occurred. If necrosis extends to involve the skin spontaneous rupture takes place.

TREATMENT—Lesions in the early stage of perianal induration without necrosis are treated medically. Management is similar to that outlined in the preceding discussion for the treatment of acute cryptitis. Should infection progress despite conservative measures surgical treatment becomes mandatory, as Allingham pointed out 70 years ago (7).

Incision for drainage—When hospitalization is necessarily delayed or impractical a simple stab incision into the abscess performed at the home, office or clinic will afford an external exit for pus under pressure thereby preventing circumferential spread of the abscess.

With 1 per cent procaine solution an intracutaneous (not subcutaneous) wheal 3 cm long is raised in the skin over the most prominent or central portion of the induration. The line of anesthesia should parallel the sphincter muscles in a circumferential direction. For this short procedure inhalation of trichlorethylene (*Trilene*) provides ample anesthesia and is preferable in apprehensive patients. The left index finger is inserted into the rectum and adjusted to support the indurated perianal mass. An exploratory stab incision is made through the anesthetized skin the scalpel being thrust cephalad and medially toward the center of the indurated region. Pus under tension is released thus affording external egress for the abscess. The skin wound is extended to measure about 3 cm and a rubber dam drain inserted into the abscess cavity. A sterilized unrolled finger cot makes a perfect drain. Wet dressings and frequent sitz baths relieve pain and lead to rapid resolution of the induration.

Definitive surgical treatment—Drainage of the acute abscess is usually the first step. After several weeks an anal fistula results which should be excised. The source of a pyogenic perianal abscess

is usually in an anal crypt. If this source of infection in the anus is removed when the abscess is drained, it is logical to assume that an anal fistula cannot occur. That this one stage stem to stern operation is feasible and practical was demonstrated by its success in 10 of 43 consecutive cases of infralevator perianal abscesses (7). The following technic was used:

With a scalpel an exploratory stab incision of the abscess is made as previously described. After the pus is evacuated the stab incision is enlarged in a direction parallel to the external sphincter muscles so that an exploratory finger can be inserted. Finger investigation determines the extent of the abscess cavity and breaks down residual fascial septa and secondary abscess pockets. Blunt scissors are introduced over the exploratory finger and the wound is enlarged to the anterior and then to the posterior limits of the abscess cavity.

Anteroposterior circumferential perianal incisions favor drainage for two reasons: (1) The fibroelastic septal insertions of the longitudinal muscle of the rectum which course radially or spoke like from the anus are cut across thereby draining individual fascial spaces (Fig. 31). (The fish mouth or hockey stick incision serves the same purpose in draining an anterior closed space infection of the finger.) (2) By its approximation to the sphincter muscles the medial wall of the abscess cavity is drawn inward and away from the fixed lateral wall. The wound edges are thereby separated so favoring drainage. In contrast, radial perianal incisions tend to close when the sphincter contracts.

A blunt curved hemostat is utilized as a probe in seeking the tract leading from the abscess cavity to the primary source of infection in the anus. This tract is found easily in most cases and the point of the hemostat can be palpated just beneath the anal epithelium. A probe pointed grooved director is substituted for the hemostat and an internal opening is sought. In some patients the probe readily emerges in an anal crypt. In many others despite careful search the probe point remains in the submucosa with only the epithelium separating the probe point from the palpating index

finger. It is probable that in these cases the abscess originated in an anal duct or in an intramuscular gland. Drainage into the anal canal was prevented by the sealing of the crypt or duct by inflammatory changes, as suggested by Gordon Watson and Dodd (2).

Obviously, one cannot leave a virulently infected sinus tract. It is my practice therefore to push the probe through the anal

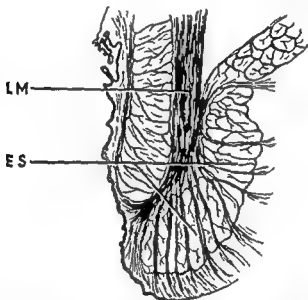


FIG 31 —Schematic drawing showing septa from longitudinal muscle (LM) coursing through sphincter muscles (ES) and areolar tissue to insert into subcutaneous fascia (Redrawn from Gorsch)

epithelium with the probe held at right angle to the course of the fibers of the external sphincter muscles. This establishes a fistulous tract between the primary opening in the anus and the distal opening in the base of the abscess.

The skin and subcutaneous tissue overlying the abscess cavity are removed and the wound edges are cut well back in order to saucerize the wound. No overhanging edges of the skin are permitted to remain. The incised tract into the anus is dealt with similarly so

that after excision of the edges the wound resembles a shallow trough. After hemostasis has been assured moistened iodoform gauze is packed into the abscess cavity and secured by adequate superficial dressings. A separate sliver of gauze is laid in the anal wound to be extruded with the first defecation. The pack is removed from the abscess on the fourth postoperative day by which time

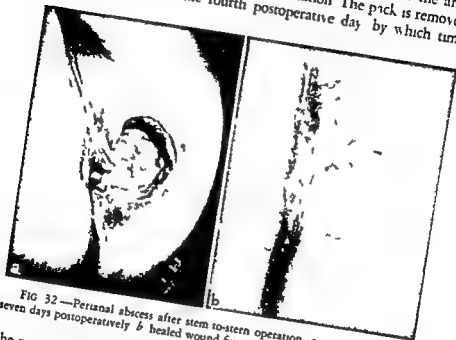


FIG 32—Perianal abscess after stem-to-stern operation. *a* saucerized abscess seven days postoperatively *b* healed wound five weeks postoperatively

the necrotic base has largely sloughed off (Fig 32) Primary stem to stern operation resulted in uncomplicated healing without fistulas in 40 of 43 cases

SUPRALEVATOR ABSCESS

Supralelevator abscesses are fortunately not common. Because of their location between the levator muscles and the peritoneal reflection these abscesses pose difficult problems in diagnosis and treatment. The large supralelevator spaces permit abscesses to attain

considerable size before the patients suffer pain. Fever, chills and prostration are early manifestations and may be present for several days before the search for the source of infection is finally directed to the rectum by the onset of discomfort there. The pelvic peritoneum, which limits the supralelevator space proximally, may become involved in the inflammatory process by direct extension. As a result, diffuse lower abdominal or pelvic pain further confuses the diagnosis of abscess of the supralelevator space. These abscesses present no visible evidence of swelling or redness. Digital examination discloses tenderness, induration and sometimes bulging on the posterior and lateral walls of the ampullary rectum well above the sphincters. Treatment is necessarily surgical. Complications occur frequently in neglected cases and are sometimes fatal. These consist of ascending phlegmons of the retroperitoneal space with septic bacteremia, portal thrombophlebitis with multiple liver abscesses and peritonitis by direct extension through the peritoneum.

Occasionally a patient complains of pain in the rectum radiating diffusely through the perineum of two or three days duration. Examination reveals no visible or palpable source of infection but the levator ani muscles are found to be very tender and spastic. No previous history of coccygodynia is elicited. In such cases the presumptive diagnosis of levator spasm with proctalgia is made. Experience has taught me that these patients bear careful observation for after two or three days perianal or high rectal induration manifests itself thereby establishing the true diagnosis of perirectal suppuration.

Supralelevator abscess is treated by external drainage through a perianal approach which is extended through the levator muscles into the supralelevator or retrorectal space. The treatment of these complicated abscesses has been thoroughly described by Courtney (8) and by Gorsch (9).

ANAL FISTULA

Although resulting from a pre-existing abscess, a fistula or fistulas are usually of long standing when patients present them.

selves for examination. They frequently do not recall or they are vague about having had an abscess. They are concerned about the annoying soiling of their underclothing and the maceration of the perianal skin caused by the discharge from the external fistulous opening.

The management of anorectal fistulas and the expected end results of treatment depend to a large extent on accuracy in diagnosis. The external orifice of the fistulous tract varies in appearance in the individual case. It is usually seen as a discharging sinus at a varying distance from the anal verge, the minute opening of which perforates an umbilicated violaceous papule of indurated skin or if of recent origin the external orifice may comprise protruding dusky granulation tissue. When multiple external openings are present the orifices may vary in appearance, the most recent presenting granulation tissue and the others showing various degrees of epithelization. Often the orifice closes temporarily in which case the external discharge is not present. In extensive honeycombing fistulas the perianal tissues and the skin of the involved area have a dusky hue and on palpation can be felt to be thickened and indurated.

Much information can be obtained by gentle and intelligent palpation. The primary anal orifice can often be felt by the inserted cotted finger as a localized tender point of induration in the upper part of the anal canal. Careful bidigital palpation can often outline the course of a simple fistulous tract by feeling a firm indurated cordlike structure coursing through the subepithelial perianal tissues at varying depths. At the turn of the century Goodsall established the fact that the fistulas with external openings in the posterior half of the perianal area will have their primary opening in the posterior half of the anus usually at or near the posterior commissure (Fig. 33). These fistulas may be simple or of the curved horseshoe type. When the external orifice is in the anterior perineum the primary opening is usually found in the anterior quadrant of the anus. Remembering and applying this rule often helps in delimiting the course of an obscure fistulous tract.

Anoscopic examination often shows the primary opening as a small dimple at the anorectal line from which a drop of pus may exude and which readily accepts the rounded point of a hooked probe. However, it is not unusual to fail to find a primary opening even after diligent search with a hooked probe under direct anoscopic vision. In such cases it sometimes helps to inject under gentle

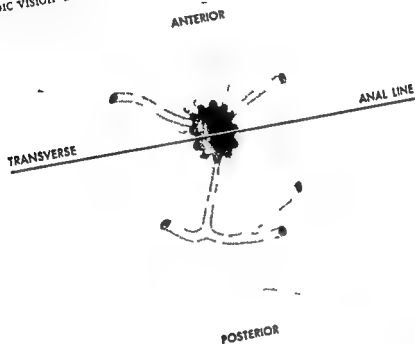


FIG 33—Goodsall's rule

pressure a sterile solution of an antiseptic dye (1 per cent solution of gentian violet) through the external orifice. A swab of cotton wool previously placed in the anus will help locate the site of the minute anal opening if one is present, and anoscopy will reveal its site as a stained point on the anal epithelium.

In high level fistulas, diagnostic roentgenography (Fig 34) with radiopaque mediums such as Lipiodol, injected through the

external orifice is necessary to orient the surgeon. It also provides valuable information regarding prognosis.

Fistula in ano must be treated surgically. In simple fistula in which the tract is easily followed by palpation or probing from the external through the internal opening, incision of the tracts over a grooved director threaded through the tract should result in cure.



FIG 34—Roentgenographic demonstration of supralevator and ischiorectal abscess with high internal opening communicating with ampullary rectum.

invariably. After incision the overhanging skin and mucosal margins are cut well back to saucerize the wound so as to provide adequate drainage. The granulation tissue on the floor of the sinus is removed by curet. The external orifice is excised and the wound is packed with moistened iodoform gauze. A separate sliver of gauze is placed in the anal portion of the wound; this is extruded with the first bowel movement in two or three days.

Fistulas of long standing though of the simple type, are thickened and fibrous and the tract is narrowed others are markedly angulated, with lateral branches coursing in all directions from the main tract In such cases it is impossible or impractical to thread the fistula on a probe Excision of the fistula by en bloc dissection is easily accomplished The external opening is dissected free from surrounding skin and subcutaneous areolar tissue A towel clip passed through the fibrous lesion near the external orifice makes an ideal retractor for putting the fistula on the stretch, which renders the fistula readily palpable directly under the skin By gradual advancing incisions of the skin over the palpable lesion the fistula is mobilized, after which the fistula is removed en bloc by scalpel dissection, with care being taken to keep close to the fibrous cord

The procedure from this point on varies If the primary opening in the anus is posterior and well below the anorectal ring (conjoined puborectalis and deep external sphincter muscles), the en bloc excision is carried through the subcutaneous sphincter and the crypt opening excised If the primary opening is in the anterior anus or at the anorectal ring in the posterior or lateral anus excision of the primary orifice and the anal part of the fistula necessarily entails excision of a considerable portion of the external sphincter muscle Excessive retraction of the cut ends of the sphincter muscles implies healing with a deformed and gaping anus, with resulting partial inability to control flatus and soft feces

Incision of the sphincteric portion of the fistulous tract is the safest procedure in an anterior fistula or a high level posterior fistula The fistulous tract having been excised from its external orifice to the lateral border of the external sphincter muscle, is held taut and the fibrous wall of the tract is carefully incised with the knife point until its lumen is exposed A probe pointed grooved director is inserted, advanced toward the anus and protruded from the internal opening With scalpel directed along the groove of the director probe, overlying tissues including sphincter muscles are incised An exteriorization of the intramuscular portion of the fistulous tract is the result of this procedure The excised lateral portion of the fistu-

lous tract is then amputated at the point where the tract enters the sphincter musculature. When the primary opening cannot be found or has closed the probe point usually can be palpated just below the anal epithelium. It is thrust through the anal epithelium thereby re-establishing the fistula and the tract is then incised. The wound edges are saucerized and the wound defects packed with iodoform gauze.

The packing in the perianal wound is allowed to remain intact for five days. Packing within the sphincter muscle however is removed in 48 hours. Since there is a four day latent period before active wound repair begins it seems illogical to disturb an open wound by removing adherent packing before the pathologic or surgically traumatized cells in the bed of the wound have been sloughed off. When removed on the fifth postoperative day the iodoform pack is not adherent and virtually falls out of the wound cavity with little or no pain. On inspection the wound is seen to be lined with early granulation tissue. The wound defect is not repacked. Further treatment consists in preventing bridging of adherent skin edges by constant separation of the approximated wound edges, removal by curet of exuberant granulation tissue when necessary, and local cleanliness which is best attained by frequent warm anal douches or sitz baths.

Staged procedures are frequently necessary in fistulas with high level primary openings such as those at or above the anorectal ring. In such cases excision of the primary opening involves division of the musculature of the anorectal ring. Wide retraction of the muscle ends follows and results in a wide anal defect with incontinence. Retraction of the sphincter muscle can be prevented by the use of a seton and a two stage procedure. After dissection and excision of the fistulous tract to the sphincter muscle border a heavy silk suture threaded on a probe is passed through the high internal opening and tied so as to encircle loosely the sphincter muscles and the medial border of the fistulectomy wound (Fig 35). The wound is then allowed to heal by granulation with the seton left intact in the remaining sphincteric portion of the fistula. After several weeks the

lateral borders of the sphincteric muscles in the region of the fistula become closely adherent to the firm block of scar tissue which has replaced the fistulectomy wound. The remaining anal and sphincteric portion of the fistula can now be incised over a grooved director passed through the tract marked by the seton. Because of the



FIG. 35.—Seton (white tape) encircling sphincters after first stage of exteriorization for chronic abscess and multiple fistulas with high level internal orifice.

firm fixation of the sphincter muscles by the fistulectomy scar, little or no retraction of the cut sphincter margins follows.

The management of extensive, ramifying fistulas with multiple tracts, high supralevator fistulas and those with secondary internal openings in the rectum is difficult even for the experienced surgeon, and should not be undertaken by the neophyte. In such cases cure demands wide excision of tissue with unavoidable resultant deformities of the perineum or anorectal canal. Clinically in cases of extensive fistulous disease in which staged multiple procedures are

planned drainage from the deep tracts ceases following the first stage in which the primary anal opening was removed with part of the fistula. It is probable that after removal of the source and feeder of infection from the anus the infected granulation tissue in the secondary tracts is gradually replaced by fibrous tissue and remains in the tissues as inactive cicatricial cords.

As in the case of abscesses, fistulas are seen as the anorectal manifestations of systemic or other diseases remote from the anal region. Fistulas complicate ulcerative colitis, regional ileitis, tuberculosis and lymphogranuloma venereum; they may also originate in osteomyelitis of the sacrum or other bony structures in the pelvis.

Anorectal fistulas should be removed surgically and must never be allowed to go on for many years without treatment. It is common for cancer to develop in long standing cutaneous fistulas (10). Smith (11) collected 42 cases of cancer developing in anal fistulas. I have encountered adenocarcinoma involving the internal orifice of an anal fistula in three cases. In all reported cases, including my own three, fistulous disease was present for many years and rarely less than 15 years. It is therefore imperative that all patients with anorectal fistulous disease be treated surgically in order to eradicate this definitely premalignant condition.

REFERENCES

1. Thiele G. H. Mineral oil and saline laxatives. *South M J* 35: 920, 1942.
2. Gordon Watson C. and Dodd H. Observations on fistula in ano in relation to intramuscular perianal glands. *Brit. J Surg* 22: 303, 1935.
3. Kratzer G. L. and Dockerty M. B. Histopathology of the anal ducts. *Surg Gynec & Obst* 84: 333, 1947.
- 3a. Kratzer G. L. Anal ducts and their clinical significance. *Am J Surg* 79: 34, 1950.
4. Hill M. R., Shyrock E. II. and ReBell F. G. Role of anal glands in the pathogenesis of anorectal disease. *JAMA* 121: 742, 1943.
5. Milligan E. T. C. Ano-rectal fistulae. *Proc Roy Soc. Med* 36: 365, 1943.
6. Whitney E. T. Infection of the anal glands. *Rev Gastroenterol* 15: 431, 1948.
7. Granet, E. Is anal fistula a necessary sequel to perianal abscess? *New York State J Med* 48: 63, 1948.
8. Courtney H. Abscesses of the deep perirectal spaces. *New York State J Med* 47: 2552, 1947.

Proctology

102

- 9 Gorsch R. V Proctologic considerations of the perineopelvic spaces /
Surg 85 556 1953
- 10 Rundle F F and Hales I B Mucoid carcinoma supervening on fist
ano Ann Surg 137 215 1953
- 11 Smith T E Relation of anorectal disease to malignancy Proc Dallas
M Soc 23 118 1957

CHAPTER SEVEN

Anal Fissure

FOR SUCH a morphologically insignificant lesion anal fissure causes pain which is overwhelming in its intensity. The intolerable pain compels the patient to seek early medical consultation in an effort to obtain relief. Consequently anal fissure is often seen in the early acute stage. True anal fissure denotes a break or defect extending through the entire thickness of the anal epidermis and often also into the underlying subcutaneous sphincter muscle. It must be differentiated from the superficial cracks in the anal epithelium and perianal skin that follow the passage of a large hard stool and the fissuration of the perianal skin often associated with pruritus ani.

PATHOGENESIS

Opinion about the exact pathogenesis of anal fissure is still divided. Several factors including trauma, the anatomic architecture of the anal region and infection are interrelated in its genesis. Anal fissures are found at the posterior commissure in approximately 90 per cent of cases, anteriorly in about 9 per cent and only rarely elsewhere around the circumference. Blaisdell (1) among others has emphasized the importance of defecatory trauma on the relatively unsupported anal verge caused by the Y shaped divergence of the superficial portion of the external sphincter muscle (see Fig. 3). This anatomic fact renders the anal outlet vulnerable to trauma by overdilatation. The subcutaneous sphincter muscle, although it completely encircles the anal outlet, is of little significance in affording

support because it stands by itself, is thin and often is fibrotic and inelastic. The posterior wall of the rectum curves forward from the hollow of the sacrum to join the anal canal which then turns sharply backward. The junction forms a right angle—the posterior anorectal angle or shelf. During defecation the force of practically the entire expulsive act is exerted on this anorectal shelf and on that posterior portion of the anal canal which has the least muscular support (2) (see Fig 2). These anatomic factors are borne out clinically by the posterior location of most fissures. Occasionally both anterior and posterior fissures occur in the same patient.

As a result of anal trauma associated with parturition anal fissure occurs more commonly in women than in men in an approximate proportion of 2:1. The usual fissure is easily seen at the anal verge when the buttocks are separated and the patient strains down. This type is most common and is classified as a low level fissure.

Proctologic investigation of a patient complaining of severe anal pain occasionally reveals a fissure in the proximal anal canal extending through the anorectal (dentate) line. Characteristically, this type occurs at the posterior commissure, extends deeply into the underlying tissues and has indurated and often undermined margins. It is probable that such a high level anal fissure (ulcers) has its origin in a posterior commissure crypt which became infected. The friable distended crypt gives way under the impact of defecation pressure ruptures and persists as a chronic indurated high level anal ulcer.

ACUTE FISSURE

Anal fissures are classified pathologically and clinically as acute or chronic. An acute fissure is seen as a deep tear through the anal margin extending into the anal canal. There is little inflammatory induration or edema at its borders. The clinical history reveals that a severe tearing burning pain occurred at the anus several days previously during the passage of a hard, overlarge fecal mass. Since that time every bowel movement has been accompanied by severe

searing pain followed for several hours by a persistent aching pain. Frequently slight bleeding with defecation is noted. Patients often refrain from defecation for several days because of their terror of the severe pain associated with and following this function.

The pain is caused by the tearing open of the inflamed sensitive fissure during passage of the fecal mass and retention in the wound of chemically irritating fecal matter and rectal secretions. Interestingly enough, persistence and extension of the lesion are aggravated through nature's attempt to splint the wound by reflex spasm of the anal sphincter muscles. The spasm is vigorous and continuous. It produces ischemia in the wound and edema of the adjacent skin. The edema is manifested as the characteristic sentinel pile associated with chronic fissure. Failure of the acute fissure to heal may be attributed primarily to ischemia of the tissues in and around the wound.

Pain and sphincter spasm are usually so intense that adequate examination is rarely possible without anesthesia. To pass an examining finger or instrument into the anus of a patient with an acute fissure is unnecessarily brutal. Topical anesthesia in the form of procaine crystals or Pontocaine solution on a thin cotton pledget laid gently into the base of the fissure for five minutes usually provides superficial anesthesia and sphincter relaxation so that examination can be comfortably completed. Even better is the anesthesia and muscle relaxation afforded by the injection of 1 per cent procaine (*see Chapter 3*).

CONSERVATIVE TREATMENT—The treatment of the early acute stage consists simply in relieving the spasm of the sphincter muscles thereby restoring normal circulatory balance. This is an office procedure and is best accomplished by injecting anesthetic solutions with prolonged action into the tissues adjacent to as well as into the sphincter muscles in the region of the fissure. In the case of a posterior commissure lesion after proper skin preparation 3 cc of 1 per cent procaine solution is slowly injected through a hypodermic needle into the perianal skin posterior and lateral to the fissure. The sphincter muscles in the posterior quadrant are infil-

support because it stands by itself, is thin, and often is fibrotic and inelastic. The posterior wall of the rectum curves forward from the hollow of the sacrum to join the anal canal, which then turns sharply backward. The junction forms a right angle—the posterior anorectal angle or shelf. During defecation the force of practically the entire expulsive act is exerted on this anorectal shelf and on that posterior portion of the anal canal which has the least muscular support (2) (see Fig 2). These anatomic factors are borne out clinically by the posterior location of most fissures. Occasionally both anterior and posterior fissures occur in the same patient.

As a result of anal trauma associated with parturition anal fissure occurs more commonly in women than in men in an approximate proportion of 2:1. The usual fissure is easily seen at the anal verge when the buttocks are separated and the patient strains down. This type is most common and is classified as a low level fissure.

Proctologic investigation of a patient complaining of severe anal pain occasionally reveals a fissure in the proximal anal canal extending through the anorectal (dentate) line. Characteristically, this type occurs at the posterior commissure, extends deeply into the underlying tissues and has indurated and often undermined margins. It is probable that such a high level anal fissure (ulcers) has its origin in a posterior commissure crypt which became infected. The friable distended crypt gives way under the impact of defecation pressure ruptures and persists as a chronic indurated high level anal ulcer.

ACUTE FISSURE

Anal fissures are classified pathologically and clinically as acute or chronic. An acute fissure is seen as a deep tear through the anal margin extending into the anal canal. There is little inflammatory induration or edema at its borders. The clinical history reveals that a severe tearing burning pain occurred at the anus several days previously during the passage of a hard overlarge fecal mass. Since that time every bowel movement has been accompanied by severe

searing pain followed for several hours by a persistent aching pain. Frequently, slight bleeding with defecation is noted. Patients often refrain from defecation for several days because of their terror of the severe pain associated with and following this function.

The pain is caused by the tearing open of the inflamed sensitive fissure during passage of the fecal mass and retention in the wound of chemically irritating fecal matter and rectal secretions. Interestingly enough, persistence and extension of the lesion are aggravated through nature's attempt to splint the wound by reflex spasm of the anal sphincter muscles. The spasm is vigorous and continuous. It produces ischemia in the wound and edema of the adjacent skin. The edema is manifested as the characteristic sentinel pile associated with chronic fissure. Failure of the acute fissure to heal may be attributed primarily to ischemia of the tissues in and around the wound.

Pain and sphincter spasm are usually so intense that adequate examination is rarely possible without anesthesia. To pass an examining finger or instrument into the anus of a patient with an acute fissure is unnecessarily brutal. Topical anesthesia, in the form of procaine crystals or Pontocaine solution on a thin cotton pledget laid gently into the base of the fissure for five minutes usually provides superficial anesthesia and sphincter relaxation so that examination can be comfortably completed. Even better is the anesthesia and muscle relaxation afforded by the injection of 1 per cent procaine (see Chapter 3).

CONSERVATIVE TREATMENT—The treatment of the early acute stage consists simply in relieving the spasm of the sphincter muscles thereby restoring normal circulatory balance. This is an office procedure and is best accomplished by injecting anesthetic solutions with prolonged action into the tissues adjacent to as well as into the sphincter muscles in the region of the fissure. In the case of a posterior commissure lesion after proper skin preparation 3 cc of 1 per cent procaine solution is slowly injected through a hypodermic needle into the perianal skin posterior and lateral to the fissure. The sphincter muscles in the posterior quadrant are infil-

trated by an additional 3 cc through an intramuscular needle inserted 2 cc from the anal verge and directed cephalad in a direction parallel to the axis of the anal canal. Anesthesia and relaxation after a two minute wait are sufficient to allow insertion of the gloved left index finger to support the fissure and the entire posterior anorectal angle. The contents of a 5 cc ampule of Rectocaine or Anucaine are then injected slowly through a moving needle into the external sphincter muscle posterior and lateral to the fissure, the inserted finger acting as a guide for the needle (see Fig. 14). Anesthesia and sphincter relaxation follow directly and digital and instrumental examination can be carried out forthwith.

These anesthetic agents are tissue irritants and infiltrate sensory nerves slowly. Particularly in patients with painful fissure the anesthesia furnished by the procaine wears off about 45 minutes after the injection and examination. At this time the long acting anesthetic has not yet penetrated sufficiently into the sensory nerves and most patients therefore have a recurrence of typical fissure pain which however, subsides in about two hours. This painful episode must be explained to the patient who being forewarned is not alarmed.

The patient is usually given 10 gr. of aspirin when the injection is completed. He is advised to take a hot sitz bath when he reaches home. The analgesia and sphincter relaxation persist for several days following injection during which time circulatory balance in and around the fissure is restored so that healing by granulation commences. Adjuvants of treatment include frequent hot sitz baths or anal douches and rectal lavage with a 2 oz. rubber bulb syringe following defecation to cleanse the fissure of irritating fecal residue. The patient is re-examined after about a week and the effect of injection is evaluated. Acute fissures often epithelize two to three weeks after this treatment.

Divulsion of the sphincter under anesthesia as a treatment for fissure is illogical and outmoded as is the use of fused silver nitrate on an acute fissure.

CHRONIC FISSURE

Patients with chronic fissures have episodes of pain during and after defecation however they are usually not nearly as intense as those during the acute stage Referred symptoms such as low back ache coccygodynia pain down the back of the thighs, and severe rectal constipation are common

Experience has shown that few anal fissures heal permanently either spontaneously or following conservative treatment When



FIG 36—Chronic anal ulcer triad hypertrophied papilla ulcer and sentinel pile Note fibrotic fibers of subcutaneous sphincter muscle exposed in ulcer base

examined a healed fissure is seen to be covered by a thin membranous friable scar which repeatedly breaks down A chronic fissure or anal ulcer is the expected result when an acute fissure fails to heal or recurs after purported healing The chronic fissure is characterized by inflamed indurated margins the base of which may expose the fibers of the subcutaneous external sphincter muscle or which may consist of thickened fibrous scar tissue As a result of the accumulation of irritating wound secretions and pathogens in the ulcer bed infection extends to the skin margin immediately external to the ulcer Edema and fibrosis of the skin take place and

give rise to the elevation commonly known as a sentinel pile. Similarly, at the internal margin of the fissure, edema and fibrosis of the associated papilla occur at the anorectal line. The typical chronic anal fissure consists of a triad composed of the hypertrophied papilla, the ulcer and the sentinel pile (Fig 36)

Secondary pathologic lesions associated with chronic anal fissure



FIG 37 —Epidermoid carcinoma originating in base of chronic anal fissure

are common. Internal hemorrhoids often adjoin the fissure on either side. Fibrosis of the pecten and fibrous infiltration of the subcutaneous sphincter muscle underlying the fissure are almost always present, resulting in a contracted inelastic anal canal.

Anal fissure as an important source of origin for a perianal abscess has not been sufficiently emphasized in the literature. It is possible that in such cases the fissure is only a stage in the genesis of the abscess, the primary site of origin being a traumatized in-

ected crypt. Approximately 10 per cent of perianal abscesses that I have operated on have been associated with a chronic fissure.

Epithelioma arising in a chronic fissure has been reported (3). In one of my patients — a 48 year old woman, pathologic examination after excision of a chronic anal ulcer revealed epidermoid carcinoma (Fig 37) arising at the anal margin. She was given adequate postoperative radiation therapy, and is still well 12 years later.

Anal chancre may grossly resemble a fissure or anal ulcer. Syphilis as a cause of anal ulcer must always be kept in mind (*see* Chapter 12).

SURGICAL TREATMENT

Properly performed excision will eradicate the ulcer and insure cure. The technic of operation must be adapted to the pathologic and anatomic genesis of the lesion. It must be designed to eradicate associated infection as well as those mechanical factors which might lead to recurrence. Simple excision of the fissure is inadequate, especially in dorsal lesions because insufficient perianal skin is removed to provide proper drainage. In such inadequately drained wounds infected secretions are retained in the wound by edema of the marginal skin thereby giving rise to inordinate postoperative pain, wound infection, delayed healing and frequent reoperations for recurrence.

An anal plastic operation such as that popularized by Gabriel (4) will insure cure with minimal morbidity. This consists of a wide triangular en bloc excision of the ulcer, sentinel pile, its associated crypts, papillae and adjacent hemorrhoids (Fig 38). Figure 39 shows the immediate postoperative wound.

Caudal or saddle block anesthesia is used; the patient is inverted and jackknifed and the buttocks are retracted by adhesive straps. Digital examination with both index fingers inserted into the rectum opposed then slowly drawn out clearly determines the presence of fibrosis in the pecten and the subcutaneous external sphincter muscle. The anorectum is again examined visually through a bivalve

speculum In a typical case we assume that the lesion consists of a posterior anal ulcer triad composing a hypertrophied papilla, a chronic anal ulcer with edematous margins, and an external sentinel pile (The treatment of fissure with complicating hemorrhoids is described in Chapter 8)

The mucosa of the sphincteric rectum 1 cm cephalad to the

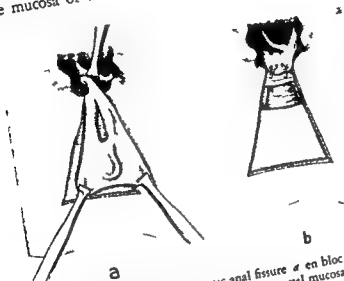


FIG 38 —Plastic operation for chronic anal fissure *a* en bloc excision of posterior anal ulcer triad *b* operation completed with rectal mucosa sutured to deep external sphincter note wide posterior drainage area and partial myotomy of subcutaneous sphincter

papilla and the anorectal line is grasped in an Allis forceps This marks the apex of our contemplated triangular excision The perianal skin 3 cm posterior to the sentinel pile is incised transversely for a distance of 4 cm This line forms the base of our triangle Lateral incisions are started at each extremity of the posterior incision and are directed toward the Allis clamp in the rectum which marks the apex For the time being the lateral incisions are carried only to the anal verge With Allis forceps on the posterior skin supplying traction a triangular skin flap previously outlined is

dissected and lifted from the subcutaneous areolar tissue and freed to the anal verge where the subcutaneous portion of the external sphincter is exposed. It will be found in most cases that the fibers



FIG 39—Granulating wounds after excision of anterior and posterior chronic anal fissures

of the exposed portion of the subcutaneous sphincter in the region of the fissure are fibrotic and inelastic.

The lateral converging incisions are now extended to complete the triangle by joining at the apex marked by the single Allis clamp. The dissection of the skin flap is then resumed, extended into the anus, thereby excising the fissure and some adjacent normal epidermis. Finally the hypertrophied papilla is dissected free and the entire triangular specimen is amputated just distal to the Allis clamp at the apex of the excised lesion. The free edge of the rectal mucosa is then sutured loosely to the exposed distal fibers of the

deep external sphincter muscle with several interrupted sutures of plain catgut

Not infrequently, the fibrotic subcutaneous external sphincter muscle is fused to the base of the anal ulcer and cannot be separated. In such cases the involved portion of the muscle is excised together with the fissure. When the fissure can be dissected free from the muscle it will be noted that, following excision of the pathologic fissure triad, the anal canal remains contracted on examination by palpation. The posterior portion of the subcutaneous sphincter muscle and fibrotic pecten remaining in the wound form a rigid muscle bar which limits mobility and dilatation of the anus. Should this muscle bar be permitted to remain, conditions favorable for reformation of anal fissure would be perpetuated. (1) Permanent cure can be assured by excising a sufficient width of fibrotic muscle so that the posterior anal orifice becomes soft and dilatable.

The following procedure is used in these patients. The subcutaneous sphincter muscle is dissected free by blunt dissection in its exposed portion. At each wound border the muscle is secured with an Allis forceps and the portion of the muscle between the clamps is excised. If arterial bleeding appears at the excised muscle borders the arteries are clamped and ligated before the Allis clamps are released. Bleeding from a muscle bundle after it has retracted from sight may be an exceedingly troublesome postoperative complication.

That the posterior quadrant of the subcutaneous sphincter muscle can be sacrificed with equanimity is supported by clinical experience and by the authoritative opinion of Milligan. (5) Writing of the subcutaneous external sphincter muscle he stated: "In defecation it should dilate. Apart from this it has no active or indispensable part in defecation. In the human being it can be cut in any way without ill effect upon defecation or continence. Continence depends on the rest of the anal musculature."

This anoplasty gives the patient freedom from both local and referred pain, a mobile dilatable anus which conforms and is supported by the Y shape of the superficial external sphincter muscle and in many instances freedom from habitual use of cathartics.

REFERENCES

- 1 Blaisdell P C. Pathogenesis of anal fissure Surg Gynec & Obst 65 672 1937
- 2 Buse L A. *Practical Proctology* (Philadelphia W B Saunders Company 1938)
- 3 Smith T E. Relationship of anorectal disease to malignancy Proc Dallas County M Soc 23 118 1937
- 4 Gabriel W B. *The Principles and Practice of Rectal Surgery* (4th ed London H K. Lewis & Co Ltd 1948)
- 5 Milligan E T C. Anorectal fistulae Proc. Roy Soc Med 36 365 1943

CHAPTER EIGHT

Hemorrhoids

HEMORRHOIDS ARE varicose veins occurring in the anorectum and originating in the plexus formed by radicals of the superior, middle and inferior hemorrhoidal veins. These tumors are accompanied by secondary inflammatory changes consisting of cellular infiltration of the overlying mucosa and anal epithelium. In general internal hemorrhoids are located under the mucosa of the sphincteric rectum proximal to the anorectal (dentate) line, external hemorrhoids are those occurring distal to this line in the anal canal or at the anal verge.

ETIOLOGY AND PATHOLOGY

The almost universal occurrence of hemorrhoids in the adult is dependent on such factors as anatomic structure, heredity, mechanical stress and infection. The veins draining the rectum—the superior hemorrhoidal, inferior mesenteric, and portal—contain no venous valves (1). In the prehistoric quadruped position of man gravity aided the return of venous blood from the rectum to the liver and right side of the heart so that the supportive aid of venous valves was not required. When man assumed the erect position the weight of this column of blood unassisted by valves established a venous pressure at the lower rectum which probably has no parallel in the body. Proof of the immense hydrostatic pressure exerted on the hemorrhoidal veins was established by Taylor and Egbert (2). Manometric readings of the pressure within a hemorrhoidal vein of

patients under saddle block anesthesia were made in the horizontal and then in the upright position. In the horizontal position the readings varied from 220 to 250 mm. of water, in the sitting position the pressure rose to between 600 and 750 mm. A further factor was emphasized by Bue (3) who pointed out that the superior hemorrhoidal veins perforate the rectal wall obliquely to enter the mesentery so that the musculature of the bowel exerts a constricting and obstructive influence on the free return of venous blood from the hemorrhoidal plexus (see Fig 5). Heredity undoubtedly is an important factor. The fact that varicose veins in the leg and in the anal canal often occur concomitantly and are frequently hereditary lends credence to this statement (4).

During defecation a large fecal bolus progressing through the rectum pushes the blood ahead of it; this retrograde milking of the veins exerts extreme pressure in the hemorrhoidal plexus. The prolonged straining at stool practiced by the chronically constipated is another mechanism favoring development of hemorrhoidal varicosities. During pregnancy and at parturition venous pressure in the pelvis is markedly exaggerated. In many women hemorrhoids develop during and after this period. Occupations in which dependent venous pressure is high have some importance in the etiology of hemorrhoids as shown by the prevalence of symptomatic hemorrhoids in truck, bus and taxi drivers, policemen, farmers, laborers and sales clerks. In constipated individuals the cathartic habit with its inordinate straining and irritating liquid stools is a potent factor favoring the development of hemorrhoids.

Excised hemorrhoids, on microscopic examination show round cell infiltration and scarring in the areolar tissue surrounding the vessels. Repeated trauma associated with the act of defecation produces breaks in the mucopithelial lining of the anorectum which allow access of infection to the vascular structures coursing immediately below the mucosa. This inflammatory involvement of the intima of the vein (phlebitis) contributes to further weakening of the vein wall and is probably the direct cause of acute thrombosis in hemorrhoids.

SYMPTOMS AND CLASSIFICATION

The patient who consults a physician for rectal disease of practically any nature states that he is troubled with hemorrhoids or piles. He complains of bleeding, burning, itching or protrusion of the piles. These symptoms are characteristic of a number of anorectal conditions among them cryptitis, fissure, polyposis, fistula, and cancer. Indeed, many of these conditions co-exist with hemorrhoids, as shown by Buie (3). Smith (5) collected 40 cases of anal cancer involving hemorrhoids and additional cases are being reported with increasing frequency. Lymphomas have been repeatedly misdiagnosed as hemorrhoids (6).

The concept that anorectal conditions occur singly must obviously be changed. Increasing experience and accuracy of clinical observations have taught us that such entities as fissures or hemorrhoids are rarely present as solitary lesions. Almost always cryptitis, papillitis and hemorrhoids co-exist. To insure permanent cure an operation for hemorrhoids must eradicate all secondary lesions.

The management of hemorrhoids depends primarily on the morphology and pathology found in the individual patient. Perhaps the most useful classification is that used at St. Mark's Hospital, London, as reported by Dodd (4). He stated:

We classify hemorrhoids into three degrees. In the first stage the veins of the anal canal are increased in number and size. During the action of defecation they are traumatized and bleed. In the second stage the internal and external hemorrhoidal veins are enlarged and the longitudinal muscle of the rectum is somewhat stretched in contrast to the normal in the first degree. This type of hemorrhoid both bleeds and presents during defecation but afterward returns spontaneously without any assistance from the patient.

The third degree of hemorrhoid consists of a large mass of both internal and external hemorrhoidal veins which is entirely outside the anal canal. The fact is that the external sphincter keeps the mass outside.

Figure 40 illustrates a third degree hemorrhoid. The St. Mark's Hospital classification of hemorrhoids is used as an aid in our selection of patients for injection or for surgery.

Although almost everyone has hemorrhoids they are usually symptomless. They give rise to symptoms when aggravated by trauma associated with bouts of diarrhea the strain required to pass an overlarge inspissated stool and often after excessive indulgence in alcohol or condiments. Large hemorrhoids of long duration, especially in the older age group are associated with mucosal



FIG 40—Third degree hemorrhoids with mucosal prolapse

prolapse and are subject to trauma when protruded in the course of each bowel action. Through repeated trauma and infection the protective mucosa overlying the varix becomes thickened, fibrotic and inelastic. It may be lacerated during defecation, the tear often extending through the thin vein wall of the distended hemorrhoid to result in an alarming spurt of blood. Fortunately bleeding is momentary during the phase of straining and dilatation. At the completion of defecation with normal contraction of the sphinc

ters and subsidence of increased venous tension, the patent vein wall is compressed and bleeding ceases. Constant repetition of these severe bleeding episodes may lead to profound secondary anemia. Superficial erosion or ulceration of the hemorrhoidal varices causes less severe bleeding. When associated with prolapse, the everted



FIG 41 —External thrombosed hemorrhoids (perianal hematomas)

hemorrhoids and even the exposed rectal mucosa bleed from their granular eroded surfaces.

Hemorrhoids are painful only when thrombosis occurs. Thrombosis of the vein with its expanding infected clot distention and edema of adjacent tissues, results in severe tension pain when it is transmitted through the sensitive somatic sensory network of the anus. External thrombosed hemorrhoids are commonly seen as beanlike tumors at the anal verge (Fig 41). In internal hemorrhoids, which are usually asymptomatic because of their location above the ano-rectal line, the proximal limit of sensitivity, thrombosis occurs

occasionally. Then the firm thrombosed, sacculated internal hemorrhoidal mass and adjacent edematous mucosa are forced down during defecation prolapsing and extruding from the anus. Increasing edema prevents spontaneous or even manual replacement so that a painful everted strangulated hemorrhoidal mass results. Such a patient presents a huge doughnut of thrombosed and sloughing hemorrhoids protruding from the anus—a disconcerting and alarming sight.

DIAGNOSIS—This is established by inspection. External prolapsed or strangulated hemorrhoids are obvious on external inspection. Internal hemorrhoids cannot ordinarily be felt by digital palpation because the varicose tumor collapses between the finger and the rectal wall but are readily seen through the anoscope. If, with the anoscope in place the patient is asked to strain the hemorrhoids swell, become more cyanotic and tend to prolapse into the lumen of the anoscope. Prolapsed rectal mucosa is distinguished from hemorrhoids by the unchanging pink color of the former even during straining. A rectal polyp, a hypertrophied papilla and simple lymphoma may sometimes be mistaken for a thrombosed internal hemorrhoid. It is worth stressing again that when rectal bleeding occurs in a patient with hemorrhoids the possibility of a neoplasm higher in the rectum or sigmoid must be ruled out—by sigmoidoscopic examination and if possible by barium enema—before treatment of the hemorrhoids is started.

ACUTE HEMORRHOIDAL DISEASE

Pain and tenderness caused by an acute external thrombotic hemorrhoid demands early treatment. What is done depends on several considerations. If the tumor mass is large, solitary and of only 1 or 2 days' duration the best interests of the patient are served by surgical excision.

SURGICAL TREATMENT

Surgical treatment can be performed at the office. This saves the patient pain and discomfort during the time required for spon-

the condition usually requires two to three weeks hereafter, elective hemorrhoidectomy should be performed in order to prevent early recurrence

CHRONIC HEMORRHOIDAL DISEASE

The excessive discomfort and pain of chronic and extensive hemorrhoids finally cause the patient to seek relief often after a long course of futile self medication with salves, suppositories, and numerous patent pile remedies. As the anorectal region is easily accessible to examination and treatment the patient afflicted with hemorrhoids expects from his physician, and is entitled to permanent cure. This can be accomplished with little loss of working time and with little pain and morbidity. Such desiderata can and should be attained. Too frequently, they are not.

SCLEROTHERAPY

The sclerosing injection treatment of hemorrhoids is attractive because it is painless, requires no loss of working time and does not entail a hospital stay. The effectiveness of this treatment is based on the fibrosis resulting from the inflammatory reaction to an irritating chemical deposited in the submucosa above the hemorrhoid or into the lumen of the hemorrhoid. The relative technical simplicity of the method makes it available to the practitioner who is not necessarily a skilled surgeon. Unfortunately permanent cure can be attained in only a small percentage of patients.

Sclerotherapy is permanently effective only when the hemorrhoids are internal, small in size and number and not associated with extensive mucosal prolapse. Such ideal cases are rarely seen clinically because at this first degree stage symptoms are few and infrequent. Sclerotherapy may be valuable in the treatment of large second degree hemorrhoidal varicosities to stop hemorrhage temporarily until operation can be performed. Injection treatment in my opinion is contraindicated and dangerous in prolapsed hemorrhoids (third degree) because irreducible prolapse, thrombosis and slough may follow the injection. Sclerotherapy has its place in allaying

symptoms caused by hemorrhoids in patients with heart disease nephritis or diabetes in psychotic or senile individuals and in all conditions in which operation is hazardous or impractical. When sclerotherapy is used patients must be examined periodically for years as recurrences or new varicosities requiring further treatment.

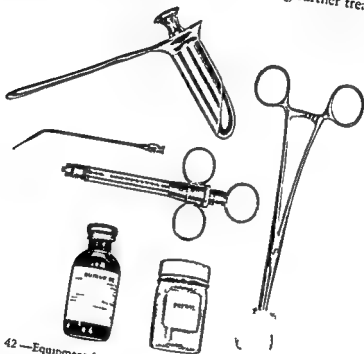


Fig 42—Equipment for sclerotherapy of internal hemorrhoids

are common. Often patients who have been thus treated for years finally submit to operation because of the intolerable discomfort resulting from increasing mucosal erosion and prolapse which can no longer be controlled by injection.

The technic of injection, indications, contraindications and dangers have been described in detail by Buie (3), Swinton (8), Terrell and Chewing (9), and Gorsch (10). A 5 per cent solution of

phenol in almond oil is a satisfactory sclerosant. This solution has a wide margin of safety because of the relatively mild but adequate inflammatory reaction following its injection into the submucosa. Treatment is administered with the patient in Sims's position. In this position hemorrhoidal swelling and mucosal prolapse are more exaggerated than in the knee-elbow position in which the hemorrhoids drain out and prolapse recedes, thereby distorting the true

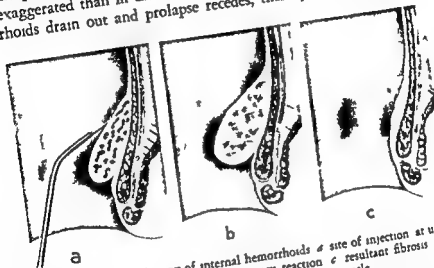


FIG 43—Sclerotherapy of internal hemorrhoids *a* site of injection at upper pole of varicosity *b* chemical inflammatory reaction *c* resultant fibrosis with obliteration of varix and adhesion of mucosa to circular muscle

pathologic extent of the lesion. A large Hirschman type of anoscope supplies excellent exposure, a 3 cc Luer Lok syringe with winged handles is very convenient, and a BD 23 gauge angled tonsil anes thesia needle affords an unobstructed view of the needle point and the site of injection (Fig 42).

The puncture point generally should be placed in the redundant mucosa directly above the internal hemorrhoidal varix (Fig 43 *a*). The mucosa is penetrated with a short jab; the needle point thus entering the loose submucosal stroma. If the needle can be freely moved under the mucosa it assuredly is placed in the submucosa and is not fixed in the underlying circular muscle. Following this important test 1 to 3 cc of the phenol-oil solution is slowly in

jected the volume used depending directly on the extent of redundancy. The mucosa must be carefully observed during the injection. The slightest surface blanching indicates improper intramucosal infiltration and injection must be stopped immediately. Just visible bulging in the area of injection indicates optimal submucosal dosage at this point. Injection is discontinued. The needle is removed and the puncture point is observed for bleeding. Should this occur tamponage with a cotton ball held in a curved artery forceps against the bleeding point for about a minute will control the bleeding. After removal of the speculum the area of injection is gently massaged with the cotted index finger. This maneuver disseminates the pooled sclerosant through the regional submucosa thereby diminishing the intensity of the chemical reaction yet increasing its over all effectiveness. On examination at the next visit, the site of injection is palpable as a firm localized induration in the submucosa extending over an area of about 2 cm. This localized induration (Fig 43 b) persists for several weeks before it is completely reabsorbed. In further treatment care must be taken not to inject into the indurated areas which are determined by palpation.

High submucosal injection accomplishes two things (1) It gives rise to proliferation of fibrous scar tissue in the submucosa which anchors the mucous membrane to the circular muscle (Fig 43 c) and so prevents further prolapse. (2) The varicose superior hemorrhoidal veins are encompassed in the submucosal fibrous tissue. As a result of this added external fibrous support further dilatation of the veins becomes impossible and with increasing scar tissue contraction the varicosities actually atrophy (Fig 43 c).

Quinuride * a 45 per cent solution of anhydrous quinine and urea adjusted to pH 2.5 with hydrochloric acid is a powerful sclerosing agent (9-11). It is an excellent sclerosant for injection directly into large internal hemorrhoidal varicosities which bleed profusely but do not prolapse. In maximum dosage of 0.5 cc its action is rapid and effective in establishing intravascular thromboses. The

tient first be examined for the presence of a constricting pecten band (Fig 45) The lubricated index fingers are inserted into the rectum hooked around the sphincter musculature, gently spread and slowly withdrawn The anal musculature gives way readily but if pec

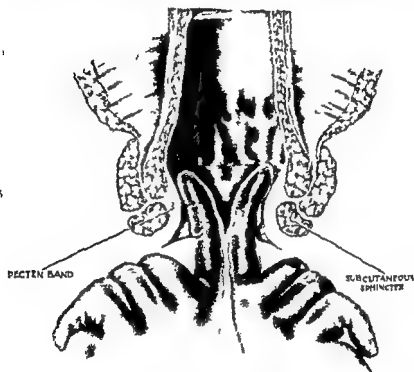


FIG. 45 — Bidigital examination of anesthetized anorectum dilatation limited by pecten band and fibrous infiltration of subcutaneous external sphincter ani.

tenosis is present spread of the opposed fingers is limited by the thin fibrous band in the subepithelium of the anal canal The presence or absence of a constricted anus radically affects the operative procedure

Ideal exposure of the anorectal operative field is attained through the use of traction sutures secured over a metal ring retractor (Fig

46) the type of which was described first by Marshall (14) and more recently by others (15-17) A simple ring retractor can be inexpensively fashioned by any ironmonger by bending a 1 cm iron rod around a pint can and welding the ends This ring should be approximately 20 cm (8 in) in diameter Four shallow equidistant

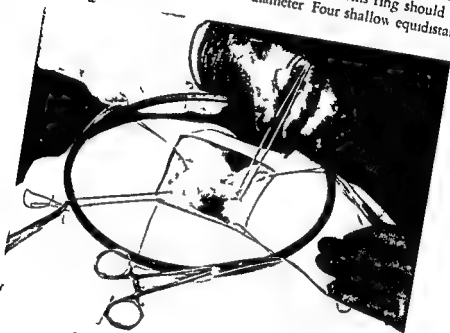


FIG 46—Exposure obtained with ring retractor

grooves cut on one surface aid in holding sutures firmly in place Traction sutures are placed in the perianal skin as illustrated The anorectum is then held open with a bivalve speculum while sutures are drawn taut adjusted in their respective grooves and secured with a Kelly clamp With lesions thus completely exposed the sequence in the procedure to be followed can now be planned The largest internal hemorrhoid is grasped in an Allis clamp and prolapsed externally Redundant rectal mucosa proximal to and

and lateral traction is exerted on the pedicle while each suture is tied very tightly on its respective side of the point of transfixion, the knots being placed on the lumen (Fig 47 c) After final inspection to make sure of absolute hemostasis the hemorrhoidal mass is excised about 1 cm distal to the ligature. A safeguard against the tie's slipping during defecation is established by leaving a considerable stump below the ligature. The stump is finally drawn

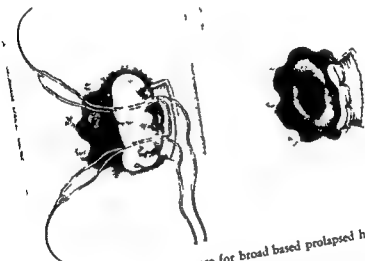


FIG 48—Goodall's triple ligature for broad based prolapsed hemorrhoids.

down to cover the defect in the perianal space and is fixed in the wound with a mattress suture of No 0 plain catgut (Fig 47 d). The remaining primary hemorrhoids are similarly excised.

When the hemorrhoidal mass is fairly broad, ligation as just described will constrict the anal canal too much. This can be avoided by utilizing the triple ligature technique originally described by Goodall (Fig 48). By use of a very long ligature threaded with two needles placed so as to divide the suture into thirds, the wide pedicle left after dissection is transfixed in a direction from the sulcus to the rectum at two points. The wide pedicle is tied in three sec-

tions with the knots on the lumen By this technic excessive narrowing of the lumen can be avoided

The ligature operation when used for relatively uncomplicated hemorrhoids is simple safe and sparing of normal tissue The strangulating transfixion ligature being firmly anchored insures against hemorrhage it sloughs off completely in eight to 10 days leaving a flat wound to heal smoothly by granulation The point of transfixion is high and well above the sensory nerves of the anus thereby minimizing pain and sphincter spasm The skin line which is cut well back from the anus provides adequate drainage and hence minimal postoperative edema A maximum of normal anal rectal epithelium is left between adjacent defects insuring adequate anal dilatibility Complete epithelization of the wounds requires two to three weeks

HEMORRHOIDECTOMY FOR EXTENSIVE AND COMPLICATED

HEMORRHOIDS—Many patients with hemorrhoids after years of self medication and perhaps several unsuccessful courses of sclerosing injections finally are referred for operative treatment of the now complicated hemorrhoids In a study of almost 14 000 patients with hemorrhoids seen at the Mayo Clinic Bue (3) found coexisting lesions which included hypertrophied papillae (35 per cent) anal fissure or ulcer (17 per cent) anal cryptitis (16 per cent) anorectal constriction (10 per cent) and anal fistula (35 per cent) Multiple lesions such as overlapping prolapsed ulcerated internal-external hemorrhoids infected crypts hypertrophied papillae anal fissure or ulcer and anal stenosis (pectenosis) are not unusual Faced with a formidable array of lesions a surgeon fearing postoperative stricture may excise only the larger varicosities But such inadequate hemorrhoidectomies soon make revision operations necessary The late complications of hemorrhoidectomy are (1) stricture (2) fissure (3) recurrent hemorrhoids (4) mucosal prolapse (5) pseudopolyps (6) excessive skin tags

Stricture—An attempt to cure a patient with markedly complicated hemorrhoids by one of the currently popular excision techniques i.e. the clamp and ligature method or the simple ligature method

just described, requires sacrifice of a considerable area of rectal mucosa and anoderm if all lesions are to be excised completely. This is unavoidable despite the most careful planning and meticulous technic at operation. In cases of multiple lesions, the loss of tissue resulting from such formidable procedures leaves only minimal bridges of intact, elastic anal epidermis and rectal mucosa. With progressive healing fibrous scar tissue proliferates and soon predominates in the anorectal canal. When healing is complete the patient has a relatively narrowed, foreshortened inelastic anorectal orifice which functionally and anatomically must be considered a stricture.

Even in the hands of the most skilful surgeons unavoidable contractures have followed excision of extensive hemorrhoids and their associated complicating lesions. Special operative techniques designed to prevent anorectal contractures in these cases have therefore been proposed by many workers (3, 18-20).

Fissure—Anal fissure or ulcer frequently develops in the patient who is left with a contracted, undilatable anus following hemorrhoidectomy. The site of the lesion usually is the posterior quadrant in or adjacent to, a recent hemorrhoidectomy scar. Repeated trauma by the forceful expulsion of a fecal mass through the narrowed orifice tears the inelastic scar, resulting in the formation of a fissure followed by infection and the subsequent persistence of the fissure as a chronic painful anal ulcer.

"Recurrent" hemorrhoids—The term recurrent, as applied to hemorrhoids, in a strict sense is a misnomer. Varicosities which have been excised cannot recur. Nevertheless, hemorrhoidal varicosities often become manifest again only a few years after a competent and complete hemorrhoidectomy. In such cases the primary hemorrhoids had probably been completely removed at operation, but varicosities of former anorectal venules because of their normal appearance at that time had been left intact. Furthermore small secondary hemorrhoids, often considered insignificant at the time of operation, are disregarded by some surgeons. These soon enlarge and become clinically significant as a source of recurrent symptoms.

Whitehead's (21) operation was devised primarily to avoid such recurrences

Mucosal prolapse—External prolapse of redundant rectal mucosa (Fig 49) with its annoying symptoms of bloody mucoid soiling skin maceration and pruritus is a fairly frequent late sequel to hemorrhoidectomy. The patient complains of these prolapsed mucosal masses as his recurrent piles. The common association of a hemorrhoidal varix and prolapse of the rectal mucosa proximal

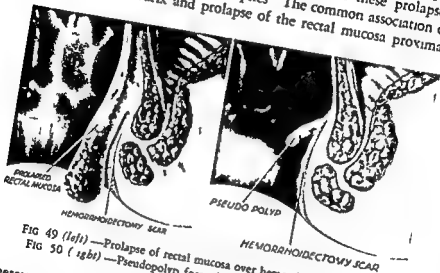


FIG 49 (left)—Prolapse of rectal mucosa over hemorrhoidectomy scar
FIG 50 (right)—Pseudopolyp formed at hemorrhoidectomy stump

thereto is well known. In a number of personally observed cases of postoperative mucosal prolapse, redundant rectal mucosa prolapsed over the cicatrix at the site of the former hemorrhoidal varix. A significant fact in these cases is that the scar tissue terminated just proximal to the anorectal line. It must be assumed that at operation the varicosities were indeed completely removed but the associated redundant rectal mucosa proximal to the hemorrhoid and in the same sagittal plane was left intact. With the passage of time the redundancy increases and eventually rectal mucosa prolapses externally over the distal hemorrhoidectomy scar. To avoid this complication, operative techniques must be used which will insure removal

mary wound the skin is again incised, this incision directed and converging toward the posterior lateral anal verge. This quadrilateral skin flap is dissected and lifted from the underlying subcutaneous fascia to expose the superficial and deep portions of the external sphincter muscles in their posterior aspects. As the anal verge is approached, the subcutaneous portion of the external

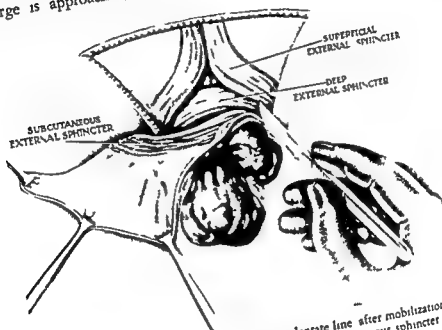


FIG 52—Dissection of quadrilateral flap to dentate line after mobilization of fissure triad adjacent hemorrhoids pecten band and subcutaneous sphincter and anoderm

sphincter muscle is exposed and is seen to be adherent to the skin and anoderm. At this point procedure varies, depending on the findings of the digital examination. In the present case, anal constriction was found. This is relieved by extending the converging skin incisions boldly through the subcutaneous sphincter muscle and pecten band. The posterior portion of the subcutaneous sphincter muscle thus becomes incorporated in the en bloc dissection of skin sentinel pile

and anal ulcer (Fig 52) The dissection has now approached the anorectal line Incision of the constricting pecten and fibrotic subcutaneous sphincter muscle mobilizes the anorectum so that by tightening the dilating traction sutures the anorectal lumen is enlarged thereby affording improved exposure

The next phase consists of a modification of Whitehead's operation By reaching high on the posterior aspect of the sphincteric rectum about 3 cm cephalad to all pathologic tissue a transverse line of rectal mucosa is grasped in three Allis clamps This normal rectal mucosa will be used to construct a new anorectal (dentate) line Gentle distal traction on the Allis clamps everts the rectal mucosa (Fig 53) thereby demonstrating that it can easily be mobilized externally to overlie the exposed border of the deep portion of the external sphincter muscle at the level of the anorectal line After mobility of the rectal mucosa is thus insured the en bloc excision is resumed The lateral skin incisions are extended to incorporate the posterolateral hemorrhoids and are continued across the anoderm and mucosa (Fig 54) Both lateral hemorrhoids are thereby included in the quadrilateral pedicle

By gentle blunt dissection with the scalpel handle the internal sphincter muscle is stripped from the submucosa of the rectum thus freeing the dissected pedicle up to the level of normal mucosa marked by the Allis clamps The dissected pedicle now contains all pathologic tissue in the posterior half of the anorectum The quadrilateral pedicle is amputated by incising the rectal mucosa just distal to the line of Allis clamps which delimits normal rectal tissue The freed mucosal edge is quickly secured with a number of Allis clamps as the anchor clamps are released (Fig 55) Two or three briskly bleeding terminal branches of the superior hemorrhoidal artery in the submucosa are clamped and tied

The free mucosal margin secured by the Allis clamps is drawn externally and is found to overlie without tension the deep portion of the external sphincter muscle at about the level of the former anorectal line It is sutured to the exposed distal free margin of the deep external sphincter muscle starting at the center and proceed

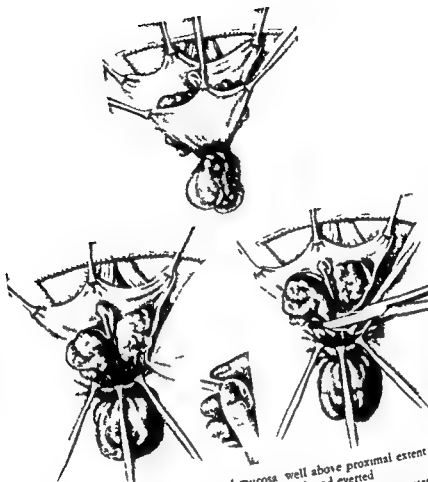


FIG 53 (*above*) —Normal rectal mucosa well above proximal extent of diseased tissue grasped by three Allis clamps secured and everted

FIG 54 (*left*) —Lateral incisions extended cephalad to include posterolateral hemorrhoids terminate just below normal mucosa outlined by Allis clamps

FIG 55 (*right*) —Amputation of quadrilateral pedicle containing all lesions : posterior half of anorectum

ing laterally (Fig 56) Interrupted fine plain gut sutures are spaced about 5 mm apart incorporating only a small portion of mucosa and muscle When the suture line is completed, venous oozing from the mucosal edge is controlled Should infection occur, sutures give way readily thereby allowing drainage into the rectal lumen We have now completed a modified Whitehead excision of

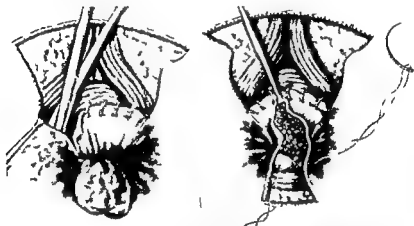


FIG 56 (*left*)—Normal rectal mucosa sutured to border of deep external sphincter and at level of dentate line subepithelial morcellation and excision of anterolateral varicosities on the left, and epithelial flap sutured to posterior perianal fascia forming pedicle graft on the right

FIG 57 (*right*)—Dissection of anterior hemorrhoidal mass pedicle transfixed through termination of fibrous longitudinal muscle of rectum

all lesions in the posterior half of the anorectum supplemented by the posterior drainage afforded by the Gabriel technic for fissurectomy The important feature of this technic is the formation of a new anorectal margin at the level of the dentate line instead of at the cutaneous verge as Whitehead directed

There now remains a large prolapsed right anterior internal external hemorrhoid as well as hemorrhoidal varicosities in both anterolateral quadrants The posterior skin and mucosal margin of one such anterolateral hemorrhoidal mass are lifted in Allis clamps

and the underlying submucosal varicose tissue is excised by morcelation and scissor dissection (Fig 56). A loose bridge of redundant mucosa, anoderm, and skin results. One or two subdermal mattress sutures are taken near its posterior edge and are secured to the subcutaneous fascia in the lateral angle of the posterior wound. The anoderm bridge is drawn flat, thus removing all redundancy and covering the lateral portion of the denuded wound with a pedicle graft (Fig 56). Dog ears of redundant skin are trimmed flat. The opposite side is similarly treated. Venous oozing from beneath the anodermal grafts is disregarded, as bleeding ceases when dressings are applied. This utilization of residual mucosa and anoderm after ablation of submucosal varicosities diminishes the area of raw wound surface and insures against a residue of excessive tags.

The remaining anterior hemorrhoid is excised by the silk ligature technic previously described (Fig 57). After final inspection of the posterior suture line tension sutures and adhesive straps are removed. The anorectum is not packed or drained. The denuded wound is covered with petrolatum gauze over which is layered a pressure dressing of plain gauze held in place by adhesive straps.

In aged patients with large prolapsing hemorrhoids, bidigital examination under anesthesia reveals that the anal sphincters dilate widely. In these patients with poor sphincter musculature the subcutaneous sphincter muscle is carefully preserved during dissection of the quadrilateral flap by peeling it away from the skin at the anal verge. By this means a firmer posterior anorectal angle is obtained, the added support of which diminishes the tendency to further postoperative prolapse. Furthermore, in these cases the posterior wound edges are not spread postoperatively as described below. The resultant bulky scar affords improved support in the weak sphincter mechanism.

Postoperative regimen—The patient receives 0.015 Gm (1/4 gr) of morphine sulfate or an equivalent drug directly on his return to bed and well before anesthesia has worn off. My experience confirms the validity of Alley's (25) statement. The maximum physiologic and pharmacologic effect of morphine is

obtained in the absence of pain. The presence of severe pain will greatly reduce or completely nullify the effects of ordinarily effective doses of the drug. Thus a small or moderate dose given before or immediately after the onset of discomfort may be more effective than a large dose given later after the pain has become severe. Hypodermic sedation for severe pain is ordered every three hours if necessary and its use is encouraged. The average patient requires 3 doses in the first 24 hours. It should be obvious that a fresh wound heals better and bleeds less when splinted as it is in a sedated, quiet patient.

Oil soluble or other local anesthetics of prolonged action are not used routinely. Except for the day of operation, discomfort at the operative site is readily controlled by sitz baths, warm moist dressings, and aspirin. The advantageous sedative effect produced by anesthetics of prolonged action is offset by the lamentable loss of awareness of impending defecation and the resultant accidents experienced by many patients, especially when ambulant. Their use is reserved for the occasional frightened patient with extremely high pain perception.

On the first postoperative day dressings are removed and the edges of the posterior wound are spread to prevent adherence and bridging. During the hospital stay, daily spreading is necessary to insure adequate drainage. A high protein, high vitamin diet is instituted and frequent sitz baths are ordered. A hydrophilic laxative is administered three times daily in a full glass of water and mineral oil once daily. A formed, firm, slippery and nonirritating evacuation results usually on the third postoperative day, after which the mineral oil is discontinued. At home the patient continues the hydrophilic colloid and maintains a full diet. Activity is not limited. For ambulatory patients the perianal douche is more cleansing and more convenient than sitz baths (*see* Chapter 4).

Most patients resume full activity after about two weeks. The wounds heal by granulation in four to six weeks despite the removal of considerable skin and anal epithelium from the posterior half of the anal and perianal regions. The wound is inspected

weekly. If exuberant granulation tissue is present in the posterior wound, it is removed with a sharp uterine curet. Procaine powder sprinkled on the wound makes this procedure painless. The index finger is passed into the rectum at each visit, the wound even if it is somewhat tight is not dilated. An anal scar, like a laparotomy scar, is swollen, indurated and tender for several months after operation. The swollen scar encroaches on the anal lumen diminishing its volume. Several months to a year after operation, the scar shrinks, blanches, and becomes pliable (26), and with this the diameter of the anorectum gradually increases. The changes are physiologic and require time. These facts must be explained to the patient for otherwise the prolonged period of postoperative observation might seem unjustified.

At the final examination six months after operation digital anoscopic and sigmoidoscopic examinations are repeated. The perianal scar at this time is soft and pliable, and the anal portion of the scar grossly resembles a normal anoderm. The rectal mucosa posteriorly is in its proper position at the site of the former dentate line and in appearance is almost indistinguishable from a normal anorectal junction.

EARLY POSTOPERATIVE COMPLICATIONS—*Urinary retention* is probably the most frequent complication of anorectal surgery and occurs particularly after hemorrhoidectomy. The common autonomic and somatic supply of the voluntary sphincter musculature at the bladder neck and at the anus via the sacral nerves is responsible for the intense spasm induced by afferent impulses originating in the painful postoperative area. This sphincter spasm in the deep urethra offsets the efforts of the detrusor muscles to effect evacuation of the bladder contents with resultant urinary retention.

Simple measures such as application of heat to the perineum and in men standing out of bed to void may succeed in overcoming retention. Should these measures be unavailing a cholinergic stimulating drug such as Urecholine 5 mg subcutaneously, may be tried. If unsuccessful the patient should be gently catheterized care being taken to avoid infection and trauma. Patients should not be allowed

to continue in distress for longer than 12 hours following operation. If the bladder is allowed to become overdilated a temporary detrusor paralysis ensues which prolongs retention and may require continued catheterization. After the Sitz baths are instituted the patient finds that the bath satisfactorily induces micturition.

In older men who have micturition difficulties 30 cc of 0.5 per cent aqueous solution of Mercurochrome introduced by catheter into the bladder at the completion of surgery produces a mild chemical stimulation of the detrusor muscle resulting in an effective expulsive effort (27).

Postoperative hemorrhage though infrequent after hemorrhoidectomy may occur as late as 10 days following operation. It is usually caused by a premature tearing free of a ligature or actual avulsion of the stump by trauma during defecation. Free bleeding may be noted directly on completion of defecation and blood continues to drip unabated. On examination the source of such bleeding is found at or near the anal verge. This type of bleeding can be controlled by manual pressure maintained for 10 minutes on a hemostatic pack (Oxycel Gelfoam) under several layers of folded gauze. Should this measure fail a suture ligature placed in the bleeding area after local block with procaine will be necessary.

More commonly massive postoperative anorectal hemorrhage arises in the internal hemorrhoidal stump proximal to the anal canal. In its early stages this is a concealed hemorrhage because little if any blood leaks externally through the sphincter closed anal canal. As blood accumulates and clots in the rectum this viscus dilates and stimulates the defecation reflex. The ensuing evacuation fills the toilet bowl with fresh blood and huge clots. Faintness, rapid thready pulse, cold sweating, pallor, and falling blood pressure—all signs of massive hemorrhage—rapidly supervene.

Treatment must be radical and immediate. If the patient is at home he must be rehospitalized preferably by ambulance. Morphine or an equivalent drug is administered to allay apprehension. The patient is typed and compatible blood is obtained for transfusion. In the operating room the posterior portions of the sphinc

ters are infiltrated with procaine-epinephrine solution to obtain relaxation. A bivalve speculum or the Newton Smith operating speculum is used to inspect the wounds in the anorectum. If a spurting vessel is found in the hemorrhoidal stump, an Allis clamp is

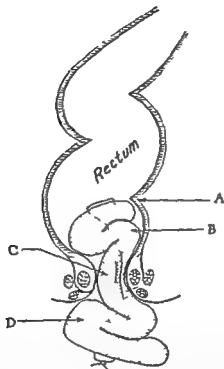


FIG 58 —Diagram for tamponade control of late postoperative hemorrhage. *A* lowest rectal valve *B* internal portion of gauze pack *C* single strip of gauze in anal canal *D* external portion of gauze pack against anal margin. (From M. E. Rolens *Am J Surg* 81:28, 1951.)

applied to achieve a wide bite around the vessel. The ordinary artery forceps will usually tear out from the partially necrotic hemorrhoidal stump. One or two suture ligatures of plain catgut will effectively control further bleeding.

It often happens that as a result of the low arterial vascular tension anemia and shock an actively bleeding artery retracts or

collapses so that by the time the patient is examined no active arterial bleeding can be found. In such cases and in those with only generalized venous oozing a firm rectal pack inserted after the rectum has been cleansed of blood and clots will insure against continued bleeding. The pack must be fashioned so that pressure is maintained on the sphincteric rectum. The rectal pack described by Rolens (28) is fashioned from a roll of vaginal packing as illustrated in Figure 58.

The packing is passed into the rectum through a tubular anoscope which is then removed. The drawstring is tightened and tied as illustrated. After 48 hours of bed rest and supportive therapy for shock including blood transfusion if clinically indicated the tied string is cut and the serum soaked packing is gently eased out of the rectum. The usual postoperative regimen is then reinstituted.

REFERENCES

1. Reuther T. F. The valves and anastomoses of the hemorrhoidal and related veins. *Am J Surg* 49:326 1940.
2. Taylor F. W. and Egbert H. L. Portal tension. *Surg Gynec & Obst* 92:64 1951.
3. Buzie L. A. *Practical Proctology* (Philadelphia: W. B. Saunders Company 1937).
4. Dodd H. In discussion on Gass O. C. and Adams J. Hemorrhoids. Etiology and pathology. *Am J Surg* 79:55 1950.
5. Smith T. E. Relationship of anorectal diseases to malignancy. *Proc Dallas County M. Soc.* 23:118 1937.
6. Granet E., Kagan M. B. and Solomon C. Lymphomas of the anorectum. *Am J Surg* 80:311 1950.
7. Cartwright E. L. The management of strangulated hemorrhoids. *Am J Digest Dis* 4:425 1937.
8. Swinton N. W. The injection treatment of hemorrhoids. *Lahey Clin Bull* 1:21 1940.
9. Terrell R. V. and Chewing C. C. Jr. Present status of injection treatment of internal hemorrhoids. *Am J Surg* 79:44 1950.
10. Gorsch R. V. Hemorrhoids. *Rev Gastroenterol* 18:859 1951.
11. Pfeifer A. C. Injection of internal hemorrhoids using 5 per cent phenol in oil. *Am J Surg* 79:49 1950.
12. Dodson J. H. and Dodson M. H. An appraisal of the injection treatment of internal hemorrhoids. *South M J* 45:347 1952.
13. Milligan E. P. C. *et al.* Surgical anatomy of the anal canal and the operative treatment of hemorrhoids. *Lancet* 2:1119 1937.
14. Marshall G. R. New anal retractor. *Northwest Med* 33:284 1934.

ters are infiltrated with procaine-epinephrine solution to obtain relaxation. A bivalve speculum or the Newton Smith operating speculum is used to inspect the wounds in the anorectum. If a spurting vessel is found in the hemorrhoidal stump an Allis clamp is

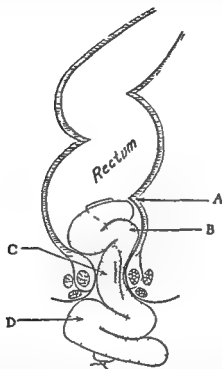


FIG 58—Diagram for tamponade control of late postoperative hemorrhage. *A* lowest rectal valve. *B* internal portion of gauze pack. *C* single strip of gauze in anal canal. *D* external portion of gauze pack against anal margin. (From M. E. Rolens *Am J Surg* 81:28, 1951.)

applied to achieve a wide bite around the vessel. The ordinary artery forceps will usually tear out from the partially necrotic hemorrhoidal stump. One or two suture ligatures of plain catgut will effectively control further bleeding.

It often happens that as a result of the low arterial vascular tension, anemia, and shock, an actively bleeding artery retracts or

collapses so that by the time the patient is examined no active arterial bleeding can be found. In such cases and in those with only generalized venous oozing a firm rectal pack inserted after the rectum has been cleansed of blood and clots will insure against continued bleeding. The pack must be fashioned so that pressure is maintained on the sphincteric rectum. The rectal pack described by Rolens (28) is fashioned from a roll of vaginal packing as illustrated in Figure 58.

The packing is passed into the rectum through a tubular anoscope which is then removed. The drawstring is tightened and tied as illustrated. After 48 hours of bed rest and supportive therapy for shock including blood transfusion if clinically indicated the tied string is cut and the serum soaked packing is gently eased out of the rectum. The usual postoperative regimen is then reinstituted.

REFERENCES

1. Reuther T. E. The valves and anastomoses of the hemorrhoidal and related veins. *Am J Surg* 49:326 1940.
2. Taylor F. W. and Egbert H. L. Portal tension. *Surg Gynec & Obst* 92:64 1951.
3. Baile L. A. *Practical Proctology* (Philadelphia W. B. Saunders Company 1937).
4. Dodd H. In discussion on Gass O. C. and Adams J. Hemorrhoids. Etiology and pathology. *Am J Surg* 79:55 1950.
5. Smith T. E. Relationship of anorectal diseases to malignancy. *Proc Dallas County M. Soc* 23:118 1937.
6. Granet E., Kagan M. B. and Solomon C. Lymphomas of the anorectum. *Am J Surg* 80:311 1950.
7. Carrwright H. L. The management of strangulated hemorrhoids. *Am J Digest Dis* 4:425 1937.
8. Swinton N. W. The injection treatment of hemorrhoids. *Lahey Clin Bull* 1:21 1940.
9. Terrell R. V. and Chewing C. C. Jr. Present status of injection treatment of internal hemorrhoids. *Am J Surg* 79:44 1950.
10. Gorsch R. V. Hemorrhoids. *Rev Gastroenterol* 18:859 1951.
11. Pfeifer A. C. Injection of internal hemorrhoids using 5 per cent phenol in oil. *Am J Surg* 79:49 1950.
12. Dodson J. H. and Dodson M. H. An appraisal of the injection treatment of internal hemorrhoids. *South M J* 45:347 1952.
13. Milligan E. P. C. *et al.* Surgical anatomy of the anal canal and the operative treatment of hemorrhoids. *Lancet* 2:1119 1937.
14. Marshall G. R. New anal retractor. *Northwest Med* 33:284 1934.

- 15 Hullsiek J R New anal retractor Minnesota Med 20 300 1937
- 16 Helfrich J R. Anal retractor Am J Surg 70 131 1945
- 17 Blaisdell P A simple pattern for a competent hemorrhoidectomy Surg. Gynec & Obst 92 140 1951
- 18 Fansler W A and Anderson J A A plastic operation for certain types of hemorrhoids J A M A 101 1064 1933
- 19 Martin E G Plastic use of skin in simple anal stricture Clinics 1944 p 195
- 20 Carmel A G Modern surgical treatment of hemorrhoids and a new rectoplasty Am J Surg 75 320 1948
- 21 Whitehead W Surgical treatment of hemorrhoids Brit M J 1 148 1882
- 22 Kantor J L Hemorrhoids—medical aspects Am J Surg 14 620 1931
- 23 Stone H B Immediate and late results of the Whitehead operation for hemorrhoids Ann Surg 58 647 1913
- 24 Granet E Anorectoplasty for extensive and complicated hemorrhoids Surgery 34 72 1953
- 25 Alley R C. Anorectal pain South M J 44 ■ 1931
- 26 Foot N C. *Pathology in Surgery* (Philadelphia J B Lippincott Company 1930)
- 27 Helfert I and Granet E. Prevention of acute urinary retention following anorectal surgical procedures Am J Surg 53 129 1941
- 28 Rolens M E Control of hemorrhage following anorectal surgery Am J Surg 81 28 1951

Benign Tumors

RECENT EMPHASIS on the value of cancer detection examinations has alerted physicians and laymen alike to the importance of the lower bowel in this program. As a result more anorectal and sigmoidal examinations by palpation and by endoscopy are being performed and lesions of the lower bowel are being discovered in ever increasing numbers. To keep our discussion practical attention will be confined to those benign tumors that occur frequently and are of clinical importance.

A useful classification of nonmalignant tumors of the anorectum and colon (Table 1) is that of Earle (1). Lymphoma is placed between the inflammatory and the neoplastic tumors because some pathologists consider lymphomas to be true tumors of the lymphatic system while others believe they are inflammatory in origin. I have added postoperative pseudopolyps to Earle's classification. Benign tumors arising in connective tissue—lipomas, fibromas, leiomyomas, and angiomas—are rare. So also are implantation cysts and desmoid tumors. Bacon discusses these rare tumors in considerable detail (2).

ANUS

Epithelial tumors which originate in the anus as papillomas are the common hypertrophied anal papillae and the less common but not unusual pseudopapillomas—*verruca acuminata* (anal warts). There is considerable evidence that the latter condition has a specific etiology and for this reason it is dealt with in Chapter 12.

TABLE 1—NONMALIGNANT TUMORS OF THE RECTUM

Epithelial	Connective tissue
Neoplastic	Lipoma
Single	Fibroma
Papilloma	Leiomyoma
Adenoma	Angioma
Carcinoid	Other tumors
Multiple	Desmoids
Acquired polyposis	Endometrioma
Familial polyposis	Implantation cysts
Verrucae	Paraffinomas
Lymphoma	
Inflammatory	
Single	
Granuloma	
Pseudopolyp (mucosal excrecence)	
Multiple	
Schistosomiasis	
Amebic granuloma	

Modified from Eadie (1)

HYPERTROPHIED PAPILLAE—The presence of hypertrophied papillae in the anus indicates long standing infection of the underlying or adjacent anal crypt. Years of infection, chronic passive congestion, and defecation trauma result in hypertrophy and elongation of normal anal papillae. Myelinated nerves, tactile corpuscles, and a rich vascular supply have been demonstrated in hypertrophied papillae. Subjective symptoms such as burning, itching, and tenesmus are probably transmitted from the anus largely through the nerve endings in the papillae.

The size and number of hypertrophied anal papillae vary. There are usually three to five papillae about 3 mm. long, although some attain a length of 10 mm. In configuration, they are conelike with the base arising at the anorectal (dentate) line. They are pink to white and consist largely of squamous epithelium. Large papillae are extruded from the anal canal with each defecation and manual replacement may be necessary. After 20 to 40 years of steady

growth they finally become huge anal protuberances (Fig 59)

The treatment of hypertrophied papillae must be planned on the basis of symptoms complications and the individual lesion. When the papillae are small multiple and associated with cryptitis or hemorrhoids removal of the papillae alone will serve no useful purpose. If symptoms are present and hemorrhoids and cryptitis are minimal conservative treatment consisting of postdefecatory rectal lavage and use of bland soothing ointments and a diet devoid

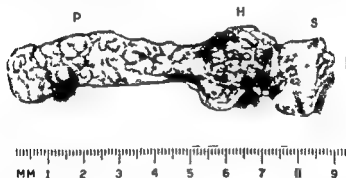


FIG 59—Papillofibroma of anus (P) excised en bloc with thrombosed external hemorrhoid (H) and perianal skin (S)

of condiments will allay subjective discomfort. Should this regimen fail an anorectoplasty to remove hemorrhoids crypts and papillae is the procedure of choice.

Large single symptomatic papillofibromas (0.5 cm or larger) which do not prolapse are best removed by diathermic snare following which the base is electrocoagulated. Injection of 5 cc of an oil soluble anesthetic into the adjacent subcutaneous areolar tissue and sphincter muscles will protect the patient from pain until the eschar sloughs and healing by granulation commences. The protruding giant papillofibromas are removed surgically with a wide skin excision for external drainage the technic being similar to that used for the excision of an anal fissure.

RECTUM SIGMOID COLON

POLYPS

Polypoid disease of the rectum and colon because of its frequent occurrence its pathophysiology, and its malignant potentiality, is an important entity. The literature on this disease has been greatly enriched in the past decade, and statistically significant facts have become available for analysis and evaluation. It is noteworthy that with minor exceptions opinion regarding frequency, pathology, and treatment is on the whole uniform.

CLASSIFICATION AND PATHOLOGY—Polypoid disease of the colorectum occurs in three distinct forms: the common adenomatous polyp, the less common villous papilloma (papillary adenoma), and the infrequent but clinically important multiple (hereditary familial) polyposis of the entire colon. The primary genetic causes of the growth of polyps are unknown. The adenomatous polyp is a tumor which arises in the epithelial cells deep in the crypts of Lieberkuhn. As it grows it becomes a compact neoplastic nodule embedded in the deep layer of the submucosa, later bulging from the surface as a minute sessile adenoma. Any tumor with a narrow base of attachment which protrudes into the lumen tends to become pedunculated from the force of peristaltic movements and the traction exerted by intestinal contents passing over its surface. The pedicle is composed of dragged-out muscularis mucosae and of submucosal tissue carrying the blood and lymph supply of the tumor and is enmeshed in normal mucous membrane. It is of considerably smaller diameter than the tumor it supports.

In large adenomas the glands arborize from the central stalk to form multilobular masses which tend to take the configuration of a mushroom (Fig. 60). In contrast the villous papillomas (Fig. 62) are soft shaggy tumors often with ill-defined edges attached by a wide base and extending over a wide area. The villous papilloma has its origin in superficial glands of the mucosal epithelium on the bowel surface. As the cells grow, branching of superficial glands takes place and the villous structure spreads widely.



FIG 60 — Malignant adenoma removed by sigmoidotomy carcinoma in situ limited to peripheral portion. No evidence of recurrence three years postoperatively.

Adjacent superficial glands join in the process perhaps because of a regional sensitization to surface neoplasms (3, 4).

The alarming frequency with which adenomatous polyps occur in the rectum and colon was not fully appreciated in the past. In two large series of autopsies on patients dying from a variety of causes grossly visible adenomatous polyps of the colon and rectum were found in 9.5 and 7 per cent respectively (5, 6). Two reviews (7, 8) of the results of sigmoidoscopy of patients without symptoms



FIG 61 —High power photomicrograph of segment of adenoma (Fig 60) showing malignant invasion of single gland with marked epithelial anaplasia



FIG 62 —Villous papilloma of sphincteric rectum prolapsed from anus

referable to the lower bowel disclosed polyps of the rectum and sigmoid in 8 per cent I found adenomatous polyps in 4.7 per cent of a similar series 16 of the polyps were sessile 5 pedunculated In a group of patients with defecatory bleeding polyps were discovered on sigmoidoscopic examination in almost 10 per cent (9) When a single polyp is found intensive and if necessary repeated examinations will frequently disclose a second or even several polyps higher up (Fig 63) The concomitant occurrence of adenoma

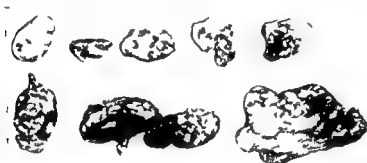


FIG 63—Multiple benign adenomas removed by cautery snare from rectosigmoid and ampullary rectum of woman aged 28 No recurrence four years post operatively

tous polyps and cancer of the lower bowel is well known Barium enema studies in patients with polyps have uncovered silent neoplastic lesions higher in the colon Accurate roentgenography including air contrast films should be performed routinely in patients with polyps

Adenomatous polyps are asymptomatic in the early stage of their growth When arborization has progressed sufficiently to form a sizable tumor trauma associated with the passage of the fecal mass may lacerate the polyp and cause bleeding Bright red blood in varying quantity usually coats the feces Pedunculated polyps low in the rectum may prolapse they may be avulsed and torn from the pedicle during defecation Villous papillomas of the rectum cause tenesmus they secrete large amounts of mucus which frequently is expelled as bloody mucoid discharges

ADENOMAS AND MALIGNANCY—In a statistically significant number of cases, cancer and benign polyps are found concurrently in the distal portion of the bowel. Lahey and Swinton (10) noted the occurrence of polyps and carcinoma in 25 per cent of all specimens of bowel obtained at operation. Many pathologists and surgeons believe that simple adenomas eventually tend to become malignant, and that the adenoma is merely a stage in the development of the malignant tumor. The highest incidence of polyps moreover, is in the fourth, fifth and sixth decades and generally parallels the age incidence of carcinoma of the bowel (5, 6).

Histologically, benign polyps have many of the characteristics of carcinoma. Such features as a bizarre architecture of structure and of individual cells, slight stratification, mitoses and increase in intensity of nuclear staining are common in both lesions. The criteria for carcinomatous transformation in polyps as established by Swinton and Warren (11) are simple and clear cut. Their views, generally accepted, are incorporated in this statement:

While the fully developed carcinoma is recognized by the anaplastic character of the cells, the irregularity of glandular structure and the invasion of not only the immediate stroma but also the adjacent intestinal wall, there are many early or transitional forms that are difficult to classify. If one accepts three important criteria of malignancy—anaplasia, irregularity of architecture and invasion—it is necessary to have at least two of these three factors present before making a diagnosis of malignant growth. It is possible for any one of these three criteria to be present without an actual malignant condition, with one exception. Definite lymphatic or intravascular invasion nearly always means a clinical malignant condition.

Anaplasia may be described as consisting of a prominence of juvenile epithelial cells with considerable variation in size and shape, with deep staining nuclei and frequent mitotic figures. The nuclei show a distortion of regular polarity so that they no longer are aligned at the base of the gland but are situated haphazardly in any position from the surface of the lumen to the basement membrane (Fig. 61).

Histologic examination of biopsy material must be performed by pathologists who are thoroughly familiar with the pathology of

the colon. For accurate diagnosis it is important that sections be taken from several portions of the adenoma or better still that the entire polyp with its pedicle be sent to the pathologist. In sessile adenomas the carcinoma may lodge in the base of the lesion, it may therefore be missed in the biopsy specimen. It is worth emphasizing that even when the biopsy specimen or the entire polyp is histologically normal the site of excision must be carefully watched for possible local tumor recurrence.

The obvious conclusions are that adenomatous polyps are common and that they should be sought for in all mature adults. When found they must be excised or destroyed by electrosurgery because of their marked tendency to become malignant (12).

TREATMENT—A high frequency machine which produces a satisfactory and stable current must be used for electrosurgery of polyps. The current may be generated through electronic tubes* or by a spark gap current†. A suction tube for removing smoke and intestinal gases is essential. It may be incorporated in the tube of the sigmoidoscope in the snare or in the ball tipped electrode. Intestinal gases contain a large proportion of hydrogen, methane and hydrogen sulfide, all of which are inflammable; the gases in the distal colon should therefore be completely removed before a spark is ignited within the lumen of the instrument. Carter (13) has reported the occurrence of major intrainestinal explosions during electrodesiccation of polyps.

The technical aspects of the electrotherapy of polyps have been ably presented by Frankfelt (14) and by Haas (15) and will not be discussed here. However, certain practical aspects concerning the treatment of sigmoidal and rectal polyps by surgical diathermy must be briefly considered. Any procedure involving shortwave electric current may be hazardous. The exact output of the machine for all settings must be known before it is used clinically. The effect of each current setting may be studied on a piece of raw beef to determine the approximate depth of penetration; this is advisable before actually treating the patient. The best setting is the lowest

Cameron Surgical Specialty Co. American Cystoscope Makers

†Bovie Electrotherm (The Liebel Flarsheim Co. Cincinnati, Ohio)

current setting which will produce the desired effect. It is always better to undertreat and, if necessary, treat soon again than to over-treat and risk bowel perforation or gross bleeding from the separation of too deep a slough.

Polyps develop a pedicle as a result of the prolonged dragging effect of peristalsis and the continuous passage of fecal matter. Pedunculated polyps are therefore generally larger and are located higher in the lower bowel than sessile polyps. Microscopic study of adequate specimens from polyps is essential. Biopsy should be performed on all sessile polyps. The biopsy forceps can be so applied that the entire visible portion of a small polyp is removed, the base and adjoining mucosa are then desiccated with a ball electrode. Most proctologists agree that pedunculated polyps should be removed by cautery snare so that they can be submitted to the pathologist in toto, except for the base of the pedicle. Large sessile polyps (5 mm or larger) must be soft and freely mobile above the submucosa in order to be desiccated with safety. If located in the rectum they can be felt with the finger in the rectosigmoid or the distal sigmoid the lesion can be palpated with the edge of the sigmoidoscope. Should the lesion prove firm to touch or fixed to the deeper coats of the bowel, the lesion may be malignant, unless its benign character has been established by adequate multiple biopsies. Electrotherapy is contraindicated in such cases.

Polyps located below the peritoneal reflection *ie*, as far as 6 cm anteriorly and laterally and 9 cm posteriorly, can be desiccated with little risk of perforation. Those situated above the peritoneal reflection but within the reach of the sigmoidoscope should be desiccated with great care. In this location it is best to use moderate current delivered by a valve machine because the depth of coagulation below the ball electrode does not exceed 15 mm.

Larger sessile polyps in the upper part of the rectum or sigmoid should be treated fractionally at intervals of approximately 10 days. Pedunculated polyps should be excised by cautery snare applied through an endoscope. The wire is looped over the polyp and with vision unobstructed the loop is contracted to engage the polyp firmly near its base close to the bowel wall. A slow coagulating

current is applied and the snare slowly closed down until the coagulated pedicle separates. The base is then desiccated with a ball electrode. Large polyps in the narrow distal sigmoid occlude the lumen so that the pedicle is hidden from view. In such cases the tip of the adenoma can be shrunk by fractional desiccation in two or three treatments. Snare removal of the residual polyp and pedicle under unobstructed direct vision is then feasible. Large pedunculated polyps have broad pedicles which contain vessels of considerable size. In these lesions excision by snare may fail to seal permanently the afferent artery because of the risk of late hemorrhage; snare removal is therefore contraindicated.

Large polyps with broad pedicles situated in the ampullary rectum under the relaxation provided by low spinal anesthesia can be grasped by the pedicle with a sponge forceps inserted through the operating speculum. Under direct vision and with careful traction polyp forceps and speculum are withdrawn from the anus. Traction procidentia of the rectum applied in this manner often delivers the polyp at the anus where it can be safely excised after firm ligation of the pedicle in sections with strangulating silk ligatures. (I have on several occasions used this method successfully to excise broad based polyps; in two cases they were located just below the rectosigmoid.) The patient with large sessile polyps or with broad pedicled polyps must be hospitalized for treatment. Such unexpected and unpredictable complications as immediate or delayed hemorrhage and accidental perforation may occur in these cases and to be prepared is to be forewarned.

Polyps beyond the easy reach of the sigmoidoscope and large sigmoidal polyps with broad inaccessible pedicles are better approached by the abdominal route. Polypoid lesions of the colon discovered by barium contrast enema and later confirmed by a repeat enema obviously must be removed by colotomy. The colonic adenoma with a long pedicle is removed in toto with a cuff of surrounding mucosa through a simple colotomy incision. Frozen section biopsy of the polyp and the pedicle provides a valuable diagnostic safety factor. Sessile adenomas or villous papillomas of the sigmoid and colon in whose depths unrecognized malignancy

may lurk, present a more serious problem. On the basis of the present safety and low mortality of segmental colon resection, Welch (16) has advocated resection as the best procedure for pre-malignant polyps of the colon. The morbidity or mortality of such resection is now, as a rule, no greater than of simple colotomy. As resection is a better operation, should cancer prove to be present in the polyp, Welch's suggestion should perhaps be generally accepted.

For practical purposes, the adenoma-cancer problem boils down to axioms long established and summarized by Bure (17), as follows. So definite is the progress of histologic metamorphosis in all polyps of the colon that, given sufficient time, it is believed that all polypoid lesions of the colon, although not actually classified as

TABLE 2—FOLLOW UP RESULTS TWO YEARS OR MORE AFTER LOCAL EXCISION OF COLORECTAL ADENOMAS CONTAINING MALIGNANT FOCI

SOURCE	CASES WITH FOCAL CANCER	LOCAL RECURRENCE AS ADENOMA	LOCAL RECURRENCE AS CANCER	CASES WITH INVASIVE CANCER	LOCAL RECURRENCE AS CANCER
McLanahan <i>et al</i> (18)	23	5	—	15	5
Castro <i>et al</i> (19)	13	—	—	—	—
Sandusky and Parsons (20)	4	—	—	—	—
Lahey (21)	33	—	—	—	—
Turnbull and Fisher (22)	20	—	—	7	—
Lockhart Mummery (23)	16	—	—	30	7

malignant growths should be treated as such and whenever possible destroyed promptly on their discovery.

Of vital interest is the ultimate fate of the patient whose rectal polyp does show malignant change either on biopsy or on histologic study after excision. Shall we be satisfied with local removal and careful follow up observations of the site or are we entirely justified in submitting our patient to a necessary but mutilating and psychologically traumatic abdominoperineal resection? This important and vexing question has been the subject of intensive investigation by a number of competent and careful observers who have reported follow up investigations at varying intervals after local removal of polyps proved to be malignant. Table 2 presents a compilation of a number of such follow up studies.

Careful study of these reports and the follow up data does not

lead to any clearcut solution of this dilemma. The opinions of these investigators may be summarized somewhat as follows:

1 Primary treatment consists of removal of the polyp with its entire pedicle. If this is impossible, multiple biopsies from various sites in the polyp become necessary, affording an opportunity for prognostic evaluation of the lesion.

2 Destruction of polyps by electrodesiccation without previous

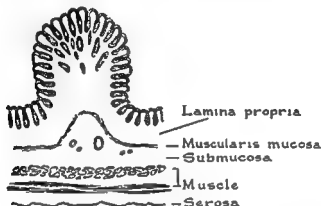


FIG 64—Diagram of carcinoma in situ arising in polyp (Figs 64 and 65 from E. R. Fisher and R. B. Turnbull Jr. *Surg. Gynec. & Obst.* 94:619, 1952.)

biopsy is to be avoided, as it affords no opportunity for microscopic study.

3 Polyps in the upper sigmoid and colon probably should be removed by local segmental resection of the bowel.

4 Rectal polyps in which histologic study shows the malignant change to be superficial (in situ) (Fig 64) and which do not invade the muscularis mucosae can be treated by local excision with safety.

5 The invasive carcinomatous polyp is one in which the malignant changes extend through the muscularis mucosae, allowing access of the malignant process to the vascular and lymphatic channels in the submucosa (Fig 65). When the base of the polyp is invaded by the carcinoma, it must be considered as frank carcinoma of the rectum.

6 Most surgeons do not perform abdominoperineal resection as a primary operation for the polyp with invasive carcinoma. The site of excision is examined at monthly intervals, radical resection of the rectum is performed if recurrence is found.

In the final analysis, the question of radical resection for inva

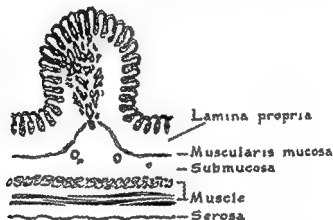


FIG 63—Diagram of invasive carcinoma arising in polyp

sive malignant rectal adenomas must be evaluated for each patient individually on the basis of the histologic picture and the patient's age condition and attitude.

VILLOUS PAPILLOMAS

Villous adenomas differ from adenomatous polyps in many respects. They occur infrequently, are large, usually solitary, and confined mainly to the rectum or distal sigmoid. Villous adenomas constituted 12.2 per cent of all polyps observed by Sunderland and Binkley (4). The transverse diameter of most of these tumors measured 4 to 6 cm. The average age of the patients was 63 years, and symptoms were frequent. Most patients have bloody, mucoid dejections due to severe tenesmus. When the tumor is low lying it is frequently extruded during defecation (see Fig 62).

Villous adenomas are slow growing, soft and velvety to the

touch and extend by a broad base over a considerable area of the rectal wall. If areas of induration are felt, these may be due to foci of carcinoma *in situ*. There is a definite tendency to recurrence after local removal of villous tumors.

These tumors even if malignant tend to remain localized and do not metastasize early. Patients with such lesions are generally elderly. It is feasible to eradicate the lesion and the adjoining segment of bowel by some type of sphincter preserving operation rather than by a radical abdominoperineal resection. Fractional surgical diathermy, radon implants and radium are useful modalities for controlling symptoms in elderly, infirm individuals whose life expectancy does not warrant the risk, morbidity, and inconvenience entailed by a resection.

MULTIPLE POLYPOSIS

Although rare, multiple (heredofamilial) polyposis is striking in its genetic, clinical, and pathologic manifestations. Approximately 350 cases have been reported in the literature. Dukes distinguished two types: a familial variety which manifests itself early in life and an acquired form appearing later, not hereditary in character. Both tend to become malignant, but the danger is greater in the familial type because the polyps are more numerous and wide spread (24). The tumors cover the entire colon and rectum and vary widely in size; the smaller ones are generally sessile, the large ones pedunculated (Fig. 66). The disease is usually limited to the large bowel and does not extend to the small intestine. It differs in this respect from the syndrome characterized by melanin spots of the oral mucosa associated with generalized intestinal polyposis, in which rare condition polyps are distributed throughout the small and the large intestine (25).

Multiple polyposis as a familial disease is handed on as a mendelian dominant transmitted by both males and females. In each of three families investigated by McCarty (26) one parent was affected (Fig. 67). Approximately half of the children of an affected

parent had polyposis, all of the children of the parents who were not affected were free of the disease

An important feature of the disease is the great propensity for cancer to develop in one or more polyps. The incidence of cancer



FIG 66 —Colon and rectum from patient with polypoid adenomatosis (From M M Ravitch and J C Handelsman Bull Johns Hopkins Hosp 88 59 1951)

occurring in multiple polyposis varies between 35 and 82 per cent. For practical purposes it must be assumed that this disease almost invariably will terminate in cancer.

In multiple polyposis, tenesmus, frequent bloody stools, abdominal pain, increasing nutritional deterioration and marked debility are characteristic and occur early. Sigmoidoscopy and barium enema studies establish the diagnosis. Once polyposis is found in a patient all members of the patient's family should be investigated. Those

who are found to be free of the disease should be kept under observation and examined sigmoidoscopically at intervals

Most patients with the disease who are alive and without cancer are below the age of 30. The majority of patients over the age of 30 if alive have cancer. Obviously, the critical change occurs between the ages of 20 and 30. The safest time for elective surgery

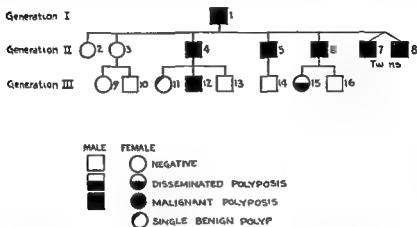


FIG 67—Familial polyposis pedigree of family group showing incidence of polyposis and carcinoma of colon and rectum (From R. T. McCarty *Am J Surg* 86:500 1953)

therefore is soon after the diagnosis is established and before the younger patients reach the middle twenties

TREATMENT—Complete surgical removal of the entire diseased colon is necessary to insure protection against otherwise inevitable carcinomatosis. It is difficult to convince the young patient and his family that a most radical surgical procedure is essential. They must be emphatically and realistically alerted to the grave consequences of procrastination. To eliminate the disease entirely, total colectomy with a permanent abdominal ileostomy is a sound procedure (28, 29). However, in view of the disagreeable consequences of a permanent ileostomy, some surgeons prefer a compromise pro-

cedure consisting of a subtotal colectomy and an ileosigmoidostomy placed just above the rectosigmoid. This is preceded or followed by thorough electrosurgical removal of all rectal polyps (27).

The type of operation to be advocated depends on the condition of the rectum. If rectal polyps are reasonably few and free from carcinoma after snare excision, colectomy with ileoproctostomy seems a logical procedure. Should a myriad of polyps be present in the rectum, a total colectomy with ileostomy is inevitable. Management based on these principles was the choice of twelve of seventeen surgeons with considerable experience in the disease who replied to a questionnaire regarding the surgical management of the disease, two surgeons favored total colectomy, and three surgeons perform conservative colectomy with ileosigmoidostomy when there is no malignancy and continuous follow up examinations are possible (30).

LYMPHOMAS

Discrete tumors consisting largely of lymphoid tissue occurring in the distal rectum or anus are being reported with increasing frequency. Simple lymphoma was defined by Ewing as a local or regional, circumscribed, chronic enlargement of lymph nodes which is self limiting and does not lead to systemic invasion.

Benign lymphoid tumors of the rectum are relatively common (31, 32). Hayes and Burr (33) collected 206 cases of benign lymphoma of the rectum from the literature and added 22 cases of their own.

Simple lymphomas occur less commonly in the sphincteric rectum and anus. We were able to find only 33 cases in the literature over a 15 year period (34). Benign lymphomas if palpable are discrete polypoid or submucosal tumors, single or multiple, varying in size from several millimeters to 3 cm in diameter. On inspection, they are seen as salmon-colored or grayish protrusions covered by intact mucosa. They occasionally form polypoid tumors on short thick pedicles. They may partially involve hemorrhoidal masses or

even infiltrate hypertrophied papillae. The true nature of the lesion can be determined only on microscopic examination.

Pathologically the lesion is composed of a single collection of overlarge, conglomerate lymph follicles often lobulated lying beneath the mucosa which may be thinned out and ulcerated in certain

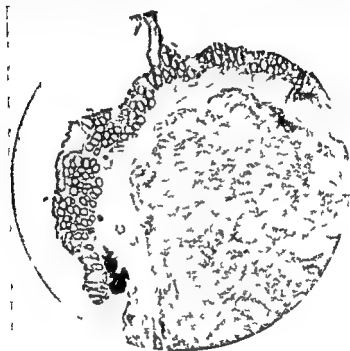


FIG 68 —Benign lymphoma of anorectum

areas. The tumor may distort the muscularis mucosae but it does not invade the muscularis propria (Fig 68). Helwig and Hansen (52) believe that because of its over all structural features diagnosis should be attempted only on the entire lesion and not on a small biopsy specimen.

Symptoms may be entirely absent the lymphoma often being found on digital rectal examination during a routine physical exam

ination Bleeding and protrusion during defecation may occur with large lesions Patients with symptoms usually have associated lesions such as hemorrhoids, anal fissure, and cryptitis

Treatment is necessarily surgical If the lesion is low in the rectum it is excised en bloc with a cuff of surrounding mucosa If high in the rectum, it is excised by cautery snare and its base then desiccated If properly excised or destroyed the lesion does not recur

FOLLICULAR LYMPHOID HYPERPLASIA—This lymphoid lesion is frequently seen in the ampulla of the rectum As a result of chronic infection involving the anorectum such as cryptitis and mild proctitis the submucosal lymphoid follicles in the ampulla of the rectum hypertrophy and are visualized endoscopically as submucosal milium tubercles about 1 mm in diameter They are distributed diffusely throughout the rectal ampulla spaced at about 5 mm intervals When the anorectal inflammatory lesions are eliminated by adequate therapy, the hyperplasia subsides Lymphoid follicular hyperplasia of the rectum lends support to Dukes and Bussey's (35) theory concerning the inflammatory genesis of lymphomas

MISCELLANEOUS BENIGN TUMORS

Benign inflammatory tumors are rare in the rectum Nonspecific granulomas occur in the cecum and other portions of the colon Amebic granulomas are also found in these locations (36) With the exception of lipomas connective tissue tumors are rare in the rectum in the colon many cases have been reported (37)

Eleomas or paraffinomas are small submucous tumors which are often palpable as discrete rounded shotty masses usually multiple located in the submucosa and extending circularly around the sphincteric rectum Pathologically they consist of fatty globules surrounded by fibrous tissue stroma They result from sclerosing injections for hemorrhoids or mucosal prolapse in which the vehicle was an oil that resisted phagocytosis Mineral oil and some forms of crude vegetable oils formerly used as a vehicle for the sclerosing agent led to the occurrence of many eleomas These oils have since

fallen into disrepute as solvents and are no longer in common use. When shotty, submucous tumors are palpated in the rectum a history of previous injection treatment for hemorrhoids can usually be obtained. The tumors require no treatment.

Endometrial tissue infiltrating the sigmoid or rectum produces a tumor visualized as a defect on x-ray examination (Fig. 69).



FIG. 69—Defect produced by endometrioma of sigmoid. *Insert* compression spot film showing intact mucosa. (Courtesy of R. H. Marshak.)

Endometriosis of the sigmoid is being increasingly recognized by the roentgenologist. The differential diagnosis of endometrioma and carcinoma of the sigmoid is difficult. Although the lesion may compromise the lumen of the bowel, mucosal invasion and ulceration rarely occur. Nevertheless, five such cases have been reported (38).

Carcinoid tumors, which are benign elsewhere in the body, frequently manifest malignant characteristics when they occur in the rectum.

REFERENCES

- 1 Earle J. Non malignant tumors of the rectum and colon *Proc Roy Soc. Med* 45 685 1952
- 2 Bacon H E *Anus Rectum Sigmoid Colon* (3d ed Philadelphia J B Lippincott Company 1948)
- 3 Dukes C E The difference between a papilloma and an adenoma of the rectum *Proc Roy Soc Med* 40 829 1947
- 4 Sunderland D A and Binkley G E Papillary adenomas of the large intestine *Cancer* 1 184 1948
- 5 Helwig L C The evolution of adenomas of the large intestine and their relation to carcinoma *Surg Gynec & Obst* 84 36 1947
- 6 Swinton N W and Haug A D The frequency of precancerous lesions in the rectum and colon *Lahey Clin. Bull* 5 84 1947
- 7 Hauch E W *et al* Adenoma of the rectum and sigmoid colon revealed by proctosigmoidoscopic examination of a group of patients free of complaints referable to the colon and rectum *Gastroenterology* 16 669 1950
- 8 Young V T Results of routine sigmoidoscopy *Rev Gastroenterol* 18 283 1951
- 9 Granet E In discussion on Young (8)
- 10 Lahey F H and Swinton N W Polyps of the colon and rectum as forerunners of cancer *Lahey Clin Bull* 7 226 1952
- 11 Swinton N W and Warren S Polyps of the colon and rectum and their relation to malignancy *JAMA* 114 1927 1939
- 12 Binkley G E *et al* Carcinoma arising in adenomas of colon and rectum *JAMA* 148 1465 1952
- 13 Carter H G Explosion in the colon during electro desiccation of polyps *Am J Surg* 84 514 1952
- 14 Frankfelt F M Endoscopic removal of polyps by electrosurgery *Proc. Roy Soc Med* 45 686 1952
- 15 Haas A C Electrodesiccation versus electrocoagulation *Am J Surg* 84 510 1952
- 16 Welch C E The treatment of polyps of the colon *Surg Gynec & Obst* 93 368 1951 (editorial)
- 17 Baile L A *Practical Proctology* (Philadelphia W B Saunders Company 1938)
- 18 McLanahan S Crove G P and Keiffer R F Conservative surgical management for certain rectal adenomas showing malignant change *JAMA* 141 822 1949
- 19 Castro A F Ault G W and Smith R S Adenomatous polyps of colon and rectum *Surg Gynec. & Obst* 92 164 1951
- 20 Sandusky W R. and Parsons J R. Jr Adenomatous polyps of colon and rectum *Ann Surg* 135 818 1952
- 21 Lahey F H In discussion on Sandusky and Parsons (20)
- 22 Turnbull R B Jr and Fisher E R Carcinoma in rectal polyps follow-up observations of 27 patients *Postgrad Med* 12 303 1952

- 23 Lockhart Mummery H E The management of benign adenomas found histologically to be early carcinomas *Proc Roy Soc. Med* 45 695 1952
- 23a Fisher E R. and Turnbull R. B Jr Malignant polyps of rectum and sigmoid Therapy based on pathologic considerations *Surg Gynec & Obst* 94 619 1952
- 24 Dukes C E. The significance of the unusual in the pathology of intestinal tumors *Ann Roy Coll Surgeons England* 1 90 1949
- 24a Ravitch M M and Handelsman J C. One stage resection of entire colon and rectum for ulcerative colitis and polypoid adenomatosis *Bull Johns Hopkins Hosp* 88 59 1951
- 25 Jeghers H McKusick V A and Katz K. H Generalized intestinal polyposis and melanin spots *New England J Med* 241 933 1949
- 26 McCarty R. T Mendelian dominant nature of familial polyposis *Am J Surg* 86 500 1953
- 27 Smith N D and Hill J R. Multiple polyps *JAMA* 148 1440 1952
- 28 Ravitch M M Total colectomy for benign conditions with a consideration of anal ileostomy with sphincter preservation *Virginia M Month* 77 55 1950
- 29 Bartlett R. W and Peck M E. Management of multiple polyposis of the colon *Surg Gynec. & Obst.* 90 547 1950
- 30 Anschuetz R. R Management of familial polyposis of the colon *Surgery* 29 532 1951
- 31 Li I Y Benign lymphoma of the rectum *Surgery* 23 814 1948
- 32 Helwig E B and Hansen J Lymphoid polyps (benign lymphoma) and malignant lymphoma of the rectum and anus *Surg Gynec & Obst* 92 233 1951
- 33 Hayes H T and Burr H B Benign lymphomas of the rectum *Am J Surg.* 84 545 1952
- 34 Granet E Kagan M B and Solomon C. Lymphomas of the anorectum *Am J Surg.* 80 311 1950
- 35 Dukes C E and Bussey H G R. The number of lymphoid follicles in the human large intestine *J Path & Bact* 29 111 1926
- 36 Rosser C. Benign tumor formation in the large bowel *South. Surgeon* 12 75 1946
- 37 Mayo C W Lipoma of the colon *Proc Staff Meet Mayo Clin* 24 9 1949
- 38 Benz E J Dockerty M B and Dixon C F Endometrioma of the sigmoid *Proc Staff Meet. Mayo Clin* 27 201 1952

Malignant Tumors

ADVANCES IN our knowledge of the basic medical sciences and their application to surgery have greatly increased the salvage rate of patients with cancer. Surgical techniques are becoming increasingly radical and more exact so that a higher resectability rate for lower bowel cancer has resulted. Patients considered inoperable a decade ago are now treated by admittedly radical procedures and so have a chance for cure.

It is not possible nor is it intended to consider here the numerous aspects of lower bowel cancer: its pathology, or its management. However, the family of a patient afflicted with rectal or colon cancer expects from the attending physician advice on the best course in striving for cure. Specifically, they wish to know about the type of contemplated operation and its expected result as regards morbidity, longevity, and the chance for ultimate cure. The physician should be prepared to discuss with the patient's family the advisability of radical operation to accomplish complete removal of the cancer and its possible extensions. He must be able to advise support and help the patient faced with the problem of an unavoidable colostomy. It is the family physician and rarely the surgeon who cares for the patient with inoperable or terminal metastatic cancer. Psychologic changes in the personality of the patient—depressed and distressed by the restriction imposed on him by his colostomy, his postoperative debility, and his inability to return to his former activities—are all problems that devolve on the family doctor. These

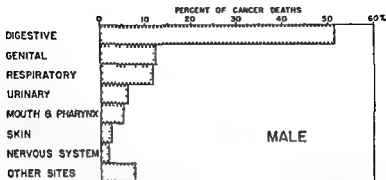


FIG. 70—Cancer death rates male (Figs 70 and 71 from American Cancer Society Statistical Research Section, Cancer Deaths by Site (1949))

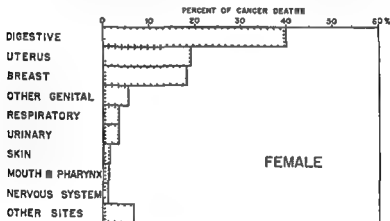


FIG. 71—Cancer death rates female

aspects of the problem of lower bowel cancer are stressed in the following discussion

The indications and contraindications for various operative procedures in specific situations are presented with reasonable brevity and operative technics as practiced in outstanding surgical clinics are summarized. Furthermore an attempt is made to evaluate the

over all results of operations for rectal cancer designed to preserve anal continence

There is undoubtedly a time when every malignant tumor is strictly a local lesion, a cancer *in situ*. If the tumor is found and eradicated at this stage cure is possible.

Table 3 and Figures 70 and 71 show graphically that the mortality from colorectal cancer in the two sexes combined is higher by far than from cancer of any other anatomic system. In males the number of deaths slightly exceeds that caused by gastric cancer; in females the death rate is only a little below that from mammary cancer.

SYMPTOMS AND DIAGNOSIS

SYMPTOMS—Recognition of gastric cancer in the early curative stage is admittedly difficult. Not so, however, is the early diagnosis of colon and rectal cancer. Gastric cancer is often symptomless at first. Colon and rectal cancers manifest symptoms while still at a stage favorable for cure. Despite this advantage there is often a lamentable delay between the onset of symptoms and admission of the patient to the hospital for definitive treatment. In one survey of 1,600 cases of rectal cancer the delay averaged $6\frac{1}{2}$ months (2). In another series reported from the Lahey Clinic (39), the delay between the appearance of the first symptoms and hospital admission for treatment averaged 14 months. Why is this so and what can be done about it?

Responsibility for delayed diagnosis of large bowel cancer originates first with the patient, and secondly with the physician whom the patient consults. Some patients refuse to consult a physician until their plight is desperate and their condition hopeless. This is especially true of those whose symptoms pertain to the terminal portion of the bowel. Some delay medical consultation because of a false sense of modesty. Others fear the discomfort and the pain which they have learned from personal experience or from hearsay to be associated with an unskillfully performed rectal examination.

TABLE 3.—CANCER DEATHS IN UNITED STATES 1949 (1)

Site	Both Sexes			Male			Female		
	No. of Deaths	%	Rate per 100,000	No. of Deaths	%	Rate per 100,000	No. of Deaths	%	Rate per 100,000
All sites	206,325	100.0	138.3	104,632	100.0	140.1	102,293	100.0	136.4
Colon and sigmoid	22,151	10.7	14.8	10,051	9.7	13.5	12,100	11.8	16.1
Rectum	10,309	5.0	6.9	5,813	5.6	7.8	4,496	4.4	6.0
Combined	32,460	15.7	21.7	15,864	15.3	21.3	16,596	16.2	22.1
Stomach	24,791	12.0	16.6	15,632	15.0	21.5	9,159	9.0	12.2
Breast	18,553	9.0	12.0	233	0.2	0.3	18,320	17.9	24.1
Lung	16,660	8.1	11.2	13,507	13.0	18.2	3,153	3.1	4.2
Cervix uterus ovaries	22,490	10.9	15.1	—	—	—	22,490	22.0	30.0
Prostate testes	12,055	5.8	8.1	12,055	11.6	16.2	—	—	—
Bladder and kidney	9,859	4.8	6.6	6,416	6.2	8.0	3,443	3.4	4.6

Still others procrastinate because they dread the possibility that their symptoms may be due to rectal cancer

The avalanche of cancer prevention propaganda by the American Cancer Society and other public health agencies has done much to make the public cancer conscious, with the result that patients are currently reporting for examination earlier

When a patient presents himself with a complaint pertaining to the function of the colon rectum, or anus, the responsibility for the diagnosis of cancer rests squarely on the physician. In a study of patients with palpable rectal cancer, Jackman (3) found that 23 per cent had already received treatment for purported conditions other than their unsuspected though palpable, cancer. Failure to diagnose cancer of the lower bowel promptly is often due to a sin of omission—the failure to obtain and evaluate an adequate history and to follow this by a careful examination. Only two minutes is required to obtain an accurate, sequential history of symptoms pertaining to the lower bowel. Important symptoms in the order of frequency of occurrence are (1) the presence of blood on the stool or following defecation (2) change in the usual pattern of bowel movement and recent excess of flatus (3) pain and tenesmus and (4) constitutional symptoms, such as anorexia, loss of weight, fatigue and weakness.

Although blood in the stool and with defecation often stems exclusively from lacerated hemorrhoids in too many instances hemorrhoids and lower bowel cancer coexist. It is unpardonable that treatment of hemorrhoids be undertaken without first ruling out, by digital and sigmoidoscopic examinations, the possibility of a carcinoma high in the rectum or sigmoid. If in the course of endoscopy blood is noted high in the rectum or sigmoid and the lesion causing it cannot be seen, careful roentgenography with contrast enema is mandatory. A patient who has had a regular pattern of bowel evacuation for years notices increasing constipation or a sense of incomplete evacuation. Sometimes these symptoms are associated with gaseous abdominal distention and an unusual increase in the amount of expelled flatus. Or, instead of constipation in

creased frequency of stools or dejections of bloody mucus may occur. These patterns may supplant each other: first constipation then loose movements and vice versa. Any change in bowel pattern in mature patients is a danger signal which calls for investigation of the large bowel for cancer.

Pain, cramps and tenesmus are later symptoms. The rectal and colonic mucosa has no somatic sensory nerve supply so that neoplasms become painful only when extensive ulceration and induration of the bowel wall take place or when distention of segments of bowel proximal to the obstructing tumor ensues. By contrast anal cancers are painful early because of the rich somatic sensory nerve supply of this region. Cancer of the right colon manifests itself by constitutional symptoms such as anemia and weakness long before a lesion becomes palpable. Cancer at this site is rarely painful and seldom obstructs. Proximal colon cancer should be suspected and ruled out in all cases of anemia especially when the result of a test for occult blood in the stools is positive.

Colon and rectal cancer have been considered a disease of maturity but a survey of the literature (4) disclosed that this is not true. For example 38 per cent of all rectal cancers seen at the Mayo Clinic during a 20 year period were in persons under the age of 30; in another report 17 per cent of 100 patients with rectal cancer were below the age of 36; in a third it was stated that cancer of the terminal bowel is as frequent among patients below the age of 45 as above it.

DIAGNOSIS—In the lower bowel 70 to 80 per cent of the cancerous lesions occur within reach of the examining finger or of endoscopic vision (Fig. 72). Abdominal examination is of little value in the diagnosis of early colon cancer. Tumors must attain considerable size before they become palpable and distention occurs only just before obstruction is complete. Adequate examination of the rectum as an essential part of a routine physical examination will disclose a considerable number of lower bowel cancers in their early curable stage. Most rectal tumors are palpable when the patient is examined in Sims's position. The supine flexed posi-

tion will often prolapse tumors in the ampullary rectum and the rectosigmoid onto the examining finger (see Fig 9)

Sigmoidoscopy is next in order and should be performed before roentgenography. It is unfortunate that the sigmoidoscope is not generally used by general practitioners or internists. When simple precautions are observed, this instrument can be safely utilized to inspect the rectum in all cases and the distal sigmoid in most. Even

- = Malignant lesion
- ⊙ = Associated polyp
- ⊕ = Assoc mult polyposis
- = Polyp
- ◐ = Double lesion

Cecum	5	}	13%	8
Ascending	1			
Transverse	6	}	13%	9
Descending	1			
Sigmoid	32	}	73	47
Rectum	25			
				35%



FIG 72 — Percentages of malignant lesions of colon and rectum which can be palpated digitally or visualized endoscopically (From U Maes and I M Estlin Ann Surg 150 1008 1949)

experienced sigmoidoscopists recognize the fact that anatomic variations such as a short mesentery and marked anterior angulation make it impossible at times to pass any distance above the rectosigmoid. In practice the barium contrast enema can be fairly accurate in demonstrating lesions above the rectosigmoid. It is of little value in revealing early rectal neoplasms. When instrumental examination of the distal sigmoid is precluded by mechanical factors or spasm of the rectosigmoid and symptoms are present, roentgenography with a contrast barium enema is in order. This procedure if correctly executed can disclose early cancer of the colon.

The bowel must be clean before the enema is given. Half an

ounce of fluffy tannic acid added to the barium mixture expedites evacuation and stimulates erection of the rugae so that exceedingly clear mucosal patterns are obtained on the postevacuation films. Air insufflation films furnish an additional diagnostic aid in revealing small neoplasms (Fig 73). Oblique views are necessary to visualize redundant portions of the sigmoid and splenic and hepatic flexures



FIG 73—Small adenocarcinoma of descending colon revealed in postevacuation film

regions usually obscured in the posteroanterior views. There is no excuse for accepting unsatisfactory films due to fecal contents, gas, or overlapping redundancies. Early curable cancer is frequently missed because of these technical failures and re-examination, though troublesome and expensive, must be ordered until diagnostically adequate films are obtained. The 24 hour film in the usual gastro intestinal series is valueless in the diagnosis of cancer of the colon. It is dangerous because a negative report may be interpreted as implying absence of disease in the colon, thus giving a false sense

of security. Too often cancer of the colon has been diagnosed by barium enema shortly after a gastrointestinal series was reported negative. Whenever gastrointestinal symptoms even suggest neoplastic disease in the large bowel a barium enema study must always precede examination with barium from above. Impaction of barium above a colonic lesion is a frequent cause of acute intestinal obstruction. It is generally agreed that with the best technical skill, the barium enema attains a diagnostic accuracy of about 90 per cent in carcinoma of the colon. In the diagnosis of premalignant polyps and early cancer of the colon, the percentage of accuracy is considerably smaller. Examinations must be repeated until the lesion responsible for gross blood in the stools has been demonstrated (5).

ANAL CANCER

Cancer infrequently originates in or adjacent to the anus. The three forms of anal epidermoid carcinoma are squamous cell epithelioma (the usual type), melanoepithelioma, and basal cell epithelioma (both rare). At the Mayo Clinic, 137 epidermoid carcinomas of the anal region were studied during a 22 year period, 127 were squamous cell epitheliomas, 7 melanoepitheliomas and 2 basal cell epitheliomas (6).

EPIDERMOID CARCINOMA *—Between 2 and 4 per cent of all lower bowel cancers are anal epidermoid carcinomas. They may occur at any age, but 90 per cent of the cases occur in the fifth, sixth and seventh decades. The tumor shows a preference for the female sex in a ratio of 3:2. A striking feature of this type of anal carcinoma is its localization high in the anal canal in women as contrasted to its equal distribution throughout the vertical axis of the canal in men. In addition the former lesions are generally highly malignant, whereas the distal lesions are of low malignancy.

Symptoms vary with the extent of the tumor and its duration. As the lesion becomes more extensive, pain and bleeding occur.

* I am indebted for much of the material on epidermoid carcinoma to the careful reviews of the entire subject by McQuarrie and Buie (6) and by Binkley (7).

early lesions are often symptomless. Pain is perhaps the most common symptom the lesion being located within the realm of somatic sensibility. Bleeding is common. In 70 per cent of the cases these cardinal symptoms appeared early. Symptoms such as constipation, change in bowel habits, loss of weight and a subjectively palpable tumor are less frequent, occurring in 10 to 30 per cent.

Unfortunately, diagnosis in the early stage may be difficult. Often these lesions are indistinguishable from benign anal ulcer, indurated hemorrhoids or superficial fistulas. Squamous cell cancer may arise in the hyperkeratotic skin associated with long standing pruritus ani. The importance of wide excision (*see Fig. 37*) of 'ironic' anal ulcers was discussed earlier. Only by careful histologic examination of all tissues removed in benign anorectal disease will it be possible to discover epidermoid carcinoma in its early, curable stage.

The lesion is not well known. In Binkley's series of 125 cases 36 patients had been treated for anal disease unrelated to cancer. Hemorrhoidectomies, fissurectomies, fistulectomies and other operations had been performed with no suspicion that cancer was present. Well advanced lesions should present no problem in diagnosis because they are fairly large, ulcerated tumors with rolled up indurated edges, are fixed and are tender. In other cases the lesion is a localized indurated mass forming the bulk of a wound which failed to heal following some form of anorectal operation. Histologic study of a biopsy specimen readily establishes the diagnosis of epidermoid cancer.

SQUAMOUS CELL EPITHELIOMA—Usually, this lesion has the appearance of an ulcer with rolled edges, sometimes it takes the form of a plaque with rolled edges and occasionally it looks like a cauliflower shaped mass or nodule. Histologically the squamous cell epithelioma consists of large pale squamous cells with formations of keratin pearls. The appearance of the lesion depends on the grade of malignancy, the higher grades being more differentiated, that is, containing a higher percentage of uniform cancer cells. Mitoses are frequent and invasiveness is high.

Epidermoid carcinoma metastasizes widely by extension through the lymphatics. Embolic spread through the inferior hemorrhoidal plexus and the systemic circulation accounts for the bizarre metastases occasionally found in this disease. Through lymphatic dissemination lesions low in the anal canal or at the anal verge eventually reach the inguinal lymph nodes. In most cases however spread is by way of the lateral lymphatic plexus in the rectal stalks to reach the lateral pelvis. These lymphatic channels follow the middle hemorrhoidal vessels to the nodes around the hypogastric artery (8). The Frei reaction may occasionally be positive, and Binkley noted the possibility of epidermoid carcinoma developing in lesions of lymphogranuloma venereum.

The over all results of treatment of squamous carcinoma of the anus are poor. From five year results reported by several authors, the average cure rate is about 35 per cent. Early, localized lesions are generally susceptible to cure. There is no uniformity of opinion as to what constitutes effective treatment. Some surgeons feel that radical surgical removal of the lesion with extensive perianal dissection and abdominoperineal excision of the anorectum offers the best chance for cure. Local excision preceded or followed by radiation therapy is advocated by others. Roux Berger and Ennuyer (9), using radiation therapy exclusively reported five year cures in 18 (35 per cent) of 51 cases. They believed that if lesions could be treated early the number of cures would be greatly increased. That this view has merit is indicated by their statement. For the eleven cases where the primary lesion did not extend beyond the limits of the anal canal at treatment, seven were cured.

Binkley's authoritative opinion on the question of therapy is logical and practical. Most small tumors situated below the mucocutaneous line are treated by radiation alone or by radiation followed by wide local excision. Large or invasive tumors are removed by abdominoperineal resection with wide dissection of the pelvic and aortic lymph nodes. High anal tumors or those extending into the rectum also must be treated by abdominoperineal excision preceded by deep radiation therapy. Radical inguinal lymph node

dissection is carried out 10 days after abdominoperineal resection. Because inguinal node metastases have occurred years after presumptive cure of the primary lesion Binkley feels that it might be advisable routinely to remove all inguinal nodes as a prophylactic measure in all cases of low anal carcinoma. In advanced cases in which operation is not feasible high voltage radiation is administered through several portals often combined with a palliative colostomy. Based on his extensive experience as a radiologist, Friedman (10) generally concurs with Binkley's views.

MELANOEPITHELIOMA—Malignant melanoma involves the anorectum more frequently than any other portion of the gastrointestinal tract. Fortunately malignant melanoma of the anus is rare, the total number of reported cases being little more than 100. The melanoblast is generally considered to originate in the embryonic neural crest and the melanoma therefore is an ectodermal tumor. It has a high metastasizing potential so that even when the parent tumor is very small metastases may already be present. Because the melanoblast possesses a great degree of cell autonomy it can survive apart from the primary tumor and reproduce a new colony of cells. The single malignant melanoblast is able to pass through the larger lymphatic channels finally to be arrested in the finer channels of the liver, lungs or bone marrow. For this reason the patient usually succumbs not to the primary tumor but to widespread metastatic lesions in the lungs, liver, brain, skeletal system and skin (11).

Symptoms are few in the early stages. The lesion may resemble a thrombosed hemorrhoid or may occur in the form of a papillomatous nodule. Diagnosis is often made from microscopic study after excision of the lesion in the course of a hemorrhoidectomy. Larger lesions appear as protruding anal tumors and may be associated with pain, tenesmus and bleeding. Inguinal nodes are often involved by the time diagnosis is established.

Treatment unfortunately is rarely curative as previously stated. Metastases are already present when the diagnosis is established. If the diagnosis is made early radical abdominoperineal excision with

wide pelvic and inguinal node dissection may occasionally result in cure. Malignant melanoma is not radiosensitive to dosages within clinical limits. McQuarrie and Buie (6) had one clinical cure after local excision of an innocuous skin tag proved on histologic examination to contain a melanoepithelioma. Braastad *et al* (12), after

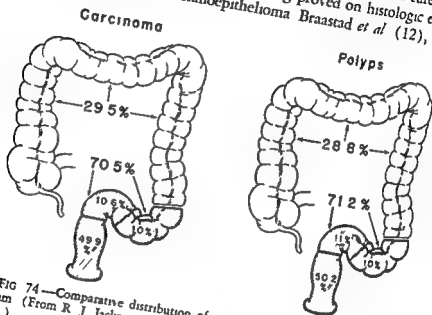


FIG 74—Comparative distribution of carcinomas and polyps in colon and rectum (From R. J. Jackman and C. W. Mayo Surg. Gynec. & Obst. 93: 32, 1951)

reviewing the literature found reports of only three cases observed long enough to be classified as clinical cures.

BASAL CELL EPITHELIOMA—This rare lesion occurs as a localized area of skin induration in the region of the anal verge and does not metastasize. Diagnostic criteria are not definite. The diagnosis is established by histologic examination after excision of the lesion or occasionally following biopsy. Gabriel Buie and Bacon all surgeons with vast experience each have seen only two cases. It is treated by wide excision preferably with the endothermy knife.

EPIDERMOID RECTAL TUMORS—Squamous cell epithelioma occurring independently in the rectum and not as an extension of

cancer primary in the anus has been authentically reported in 11 instances (13) Squamous cells in the rectum are thought to originate in anaplasia of the columnar epithelial cells of the rectum

Symptoms are similar to those caused by adenocarcinoma of the rectum Treatment necessarily consists of radical abdominoperineal excision with dissection of lateral pelvic nodes The prognosis is bad as judged by the low survival rate of the reported cases

ADENOCARCINOMA OF COLON SIGMOID AND RECTUM

Cancer of the lower bowel and rectum originates from columnar cells of the mucosa and is classified as adenocarcinoma For its genetic relationship to adenomas see Chapter 9 The adenoma-carcinoma sequence is well established by the close parallelism of the lesions in location, similarity of age incidence and frequency of their occurrence together (Fig 74)

PATHOLOGY

Adenocarcinoma is usually classified as medullary colloid and scirrhous The gross pathologic characteristics of adenocarcinoma of the rectum or colon can be summarized as follows

- 1 Marked induration of the affected tissues due to the density of cellular infiltration Lesions may protrude into the lumen as large polypoid cauliflower like tumors More characteristically the tumor is more or less circular with a polypoid indurated rolled up border and a central crater like scirrhous ulcer

- 2 Cicatricial characteristics of some large bowel carcinomas cause surrounding tissues to be drawn toward the scirrhous tumor resulting finally in an annular napkin ring stricture

- 3 Degenerative changes in the tumor are common They are manifested by areas of central sloughing in some cases and by degeneration of formed elements in others The latter is caused by overgrowth of mucinoid cells as seen characteristically in colloid carcinomas

The important histologic characteristics are (1) overgrowth of

epithelial cells (2) atypical quality of cells for example, hypertrophy, increase in nuclear chromatin, abundance of mitoses and anaplasia, (3) advancement of proliferating epithelium beyond normal limits (4) local invasive and destructive properties (5)

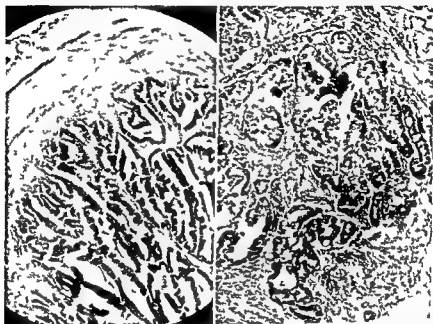


FIG 75 (left) —Grade I adenocarcinoma of rectum. Acini are well defined and dedifferentiation is slight (Figs 75-79 courtesy of Dr A. C. Broders Sr and the Scott and White Clinic Temple Tex.)

FIG 76 (right) —Grade II adenocarcinoma of rectum. Acini are not well defined and cells are about equally differentiated and dedifferentiated.

desmoplastic properties of carcinoma cells resulting in infiltration by fibroblasts plasma cells lymphocytes and blood capillaries (6) loss of nuclear polarity and (7) power to spread by metastasis.

CLASSIFICATION —Histologic study of a cancerous lesion establishes an index of its malignancy and aids in prognosis. Broders (14) classified tumors on the basis of the differentiation of their cellular elements into grades I through IV (Figs 75-79).

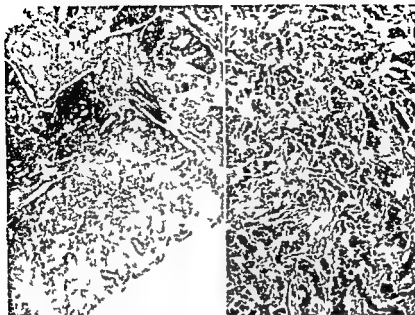


FIG 77 (*left*) —Grade III adenocarcinoma of rectum. Acini are barely defined and cells are largely dedifferentiated.

FIG 78 (*right*) —Grade IV adenocarcinoma of rectum. Cells are completely dedifferentiated.

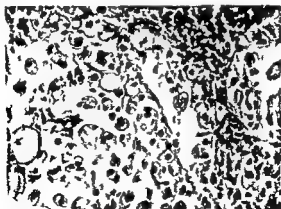


FIG 79 —Grade IV signet ring mucinous adenocarcinoma of rectum.

Broders and associates (15), in a review of histologic sections from colorectal carcinomas give the following figures

	No of Cases	Grade I	Grade II	Grade III	Grade IV
1928-40	1 610	16.4	52.5	23.7	7.4
1952	667	29.3	47.1	21.1	2.2

These figures show that most cases that are diagnosed at biopsy or at operation are already grade II or III. The current trend toward

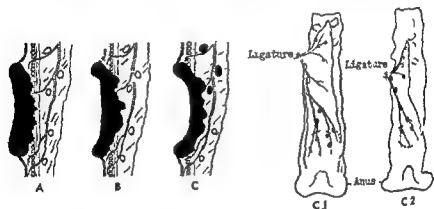


FIG 80—Dukes' classification of malignancy based on extent of spread. Grade A growth limited to wall of rectum grade B extension of growth in extrarectal tissues but no metastases in regional lymph nodes grade C, metastases to lymph nodes grade C, metastases to regional nodes grade C₁ extensive lymphatic spread (From C. E. Dukes *Am J Surg* 79:66 1950)

earlier diagnosis of bowel cancer is clearly indicated by the increased percentage of grade I cases in the 1952 series

Dukes (16) on the basis of gross examination of the excised specimen, classified tumors according to penetration of the bowel wall and the presence of metastases into grades A, B, and C, with grade C further subdivided into grades C₁ and C₂ (Fig 80)

According to Dukes at operation about 15 per cent of rectal cancers are found to be group A. The prognosis in this group is excellent as 80 to 90 per cent of the patients survive five or more years. About 35 per cent fall into group B; prognosis is relatively

good 60 to 70 per cent of the group surviving for five years after adequate excision. In the remaining 50 per cent comprising groups C₁ and C the prospect is far less satisfactory.

METHOD OF SPREAD

The opportunity to effect a cure in cancer occurs only once—at the time of the first surgical attack. If at this time the entire lesion and its metastases can be removed the patient's chance for cure is excellent.

Cancer of the colon and rectum spreads by three routes by local mural penetration by vascular channels and via the lymphatics. Lower bowel cancers are of relatively slow growth depending somewhat on the histologic grade. The tumor generally spreads more rapidly in the submucosa the circular and longitudinal muscles for a time delay extension of the growth in depth.

At the Dukes stage A the tumor rarely metastasizes to vascular or lymphatic channels. Once the tumor has penetrated the muscle barrier vascular and lymphatic spread proceeds rapidly. By contiguity the tumor spreads to adjacent structures—the ileum bladder ovaries and uterus. In general large cauliflower like tumors which grow into the lumen have low penetrating potentiality and are of low grade malignancy histologically. The small deeply ulcerating tumors penetrate the muscle barrier rapidly and metastasize widely. The annular carcinomas of the colon are the end result of prolonged growth at operation it is usually found that they have already metastasized. Grinnell (17) has shown that although at the time of discovery rectal carcinomas are generally smaller than are colon tumors the five year survival rate of patients with small rectal cancers is lower than of those with colon neoplasms of corresponding size. The reason may be that the extensive network of vascular and lymphatic channels in the perirectal pelvic region makes possible widespread metastases of rectal cancer.

LOCAL AND MURAL SPREAD—Retrograde or distal lymphatic spread of sigmoidal or rectal cancer is not common. However Connell and Rottino (18) pointed out that spread through the bowel

wall itself probably occurs frequently. By a study of serial sections, they found carcinoma in the submucosal lymph channels or in the submucosal stroma (Fig 81) as low as 7 cm from the lower margin of the tumor in four of eight specimens of Dukes grade C carci-

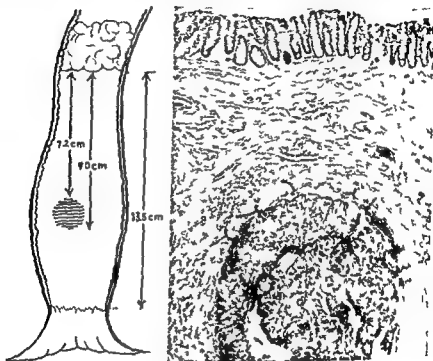


FIG 81—Metastatic intramural nodule 7 cm distal to adenocarcinoma of rectum (Figs 81 and 82 from E A Quer *et al* Surg Gynec & Obst. 96 24 1953)

nomas. Their study points out the importance of wide resection of bowel below and above cancerous lesions of the large bowel and the probable futility of operations other than abdominoperineal resection in most rectal cancers.

VASCULAR SPREAD—Sunderland (19) in a histologic study of 210 cases of rectal and sigmoidal cancer, found vein invasion in 27.6 per cent. A similar study by Grinnell (17) confirmed Sunder-

land's findings local vascular channels were invaded in 36 per cent of the cases of rectal cancer studied (Fig 82) Vein invasion was found in 4 per cent of grade A cases in 25 per cent of grade B cases and in 49 per cent of grade C cases invasion varied directly with the grade of malignancy as determined histologically

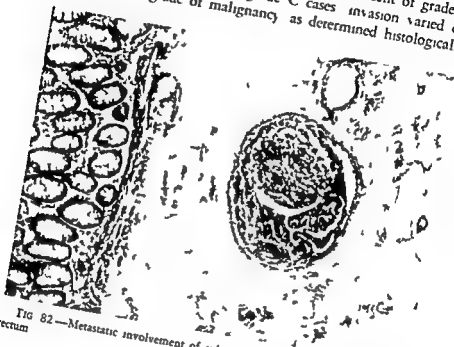


FIG 82—Metastatic involvement of submucosal vein in adenocarcinoma of rectum

(Broders) or by depth of penetration (Dukes) Distant visceral metastases have been reported in 70 to 90 per cent of cases of rectal carcinoma with vascular invasion

LYMPHATIC SPREAD—The lymphatic spread of cancer of the lower bowel and its significance in the surgical treatment and in prognosis of colorectal cancer is well known After an intensive study of cleared specimens (Fig 83) Gilchrist (20) established the fundamental fact that lymphatic spread of colorectal carcinoma is primarily embolic and its direction cephalad The node in which the

emboli lodges prevents further proximal spread until it is completely overwhelmed by carcinoma. Further embolic spread is through the lateral collateral channels making spread from one node to another uncommon, at least while the lesions are still operable. Thus the

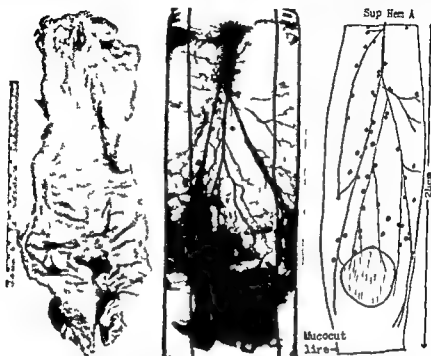


FIG 83 —Determination of lymphatic spread by study of cleared specimen and charting of lymph nodes. Left specimen of rectal carcinoma center cleared specimen right diagram showing involved nodes (solid black dots) (From R. L. Gilchrist and V. C. David Ann Surg 108 621 1938)

finding of a group of involved nodes during surgery does not mean that a case is hopeless. It does indicate the need for the widest possible resection of lymph nodes draining the area of the carcinoma.

In Grinnell's series node metastases were present in 43 per cent of the colon cases and 53 per cent of the rectal cases. He found that high grade tumors and deep mural penetration affect lymphatic metastasis directly just as they do vascular spread. It is to be ex-

pected that the greater the number of node metastases the poorer the prognosis as may be clearly seen from Table 4

It has been demonstrated that the lymphatic spread of cancer of the terminal rectum at or below the second valve of Houston proceeds laterally along the middle hemorrhoidal vessels in the lateral ligaments to drain into the lymphatic nodes on the hypogastric artery (Quenu Gerota nodes) Furthermore lymphatic spread in low rectal cancer extends concurrently along the superior hemor-

TABLE 4—RESULTS OF TREATMENT OF CANCER OF COLON AND RECTUM IN 133 CASES CLASSIFIED BY GRADE OF INVOLVEMENT (40)

INVOLVEMENT	No. of Cases	5 yr. Survival %
Vein invasion		
Absent	82	76
Present	51	37
Lymph node metastases		
Absent	64	83
Present (Grade C)	69	41
Grade C ₁	47	53
Grade C	22	18
Mural Penetration		
Grade A	17	94
Grade B	47	79
Grade C	69	41

rhoidal vessels as has been described Lesions of the upper rectum and rectosigmoid which are cephalad and remote from Kohlfrausch's fold do not metastasize laterally to the hypogastric nodes but extend only superiorly toward the aortic nodes

DIAGNOSIS BY BIOPSY

In the colon the only objective evidence of malignancy is presence or absence of a palpable mass and roentgen demonstration of the morphology of the lesion In rectal and sigmoidal lesions however not only palpable but visual and histologic information can be obtained Biopsy provides the histologic data so important in planning the surgical procedure and in prognosis Usually it can be performed in the office The lesion is visualized sigmoidoscopically and specimens are obtained by punch biopsy from three or four

sites in the more indurated portions of the tumor mass. Should profuse bleeding occur, a cotton sponge held against the bleeding area will as a rule control the bleeding. If it persists, electrocoagulation must be used. Diagnostic biopsy of extremely vascular lesions should be deferred until the patient is hospitalized for preoperative preparation.

It is often difficult to pass the sigmoidoscope full length into the sigmoid (see Chapter 2). Not infrequently bloody mucus is

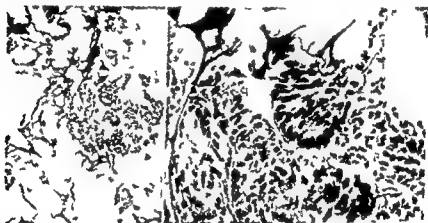


FIG 84—*Left* sponge biopsy from adenocarcinoma of rectum note particles of cancer tissue embedded in interstices of sponge. *Right* high power magnification of central section showing atypical acinus formed by large darkly stained irregular tumor cells (From S. A. Gladstone *Cancer* 2:604, 1949.)

found in the ampullary rectum during sigmoidoscopy, raising the suspicion of the presence of a lesion above the visible level. As the sigmoidoscope is advanced the lumen of the distal sigmoid is found to be occluded proximally by markedly edematous prolapsing mucosa. Attempts to manipulate the instrument past this block are defeated by the rigidity of the sigmoid above the mucosal barrier; nor will insufflation open the lumen. Manipulation increases the blood flow from above. Though invisible, the presence of a lesion just above the mucosal barrier seems beyond doubt. It is impossible to obtain a specimen by biopsy in such cases, but a specimen can be

secured by means of a sponge (24). A small Gelfoam sponge in a sponge holder can be passed through the narrow sigmoidal lumen and rubbed against the indurated but invisible mass (Fig. 84). Usually the provisional diagnosis of neoplasm can be confirmed by roentgenogram. In some clinics material for smear diagnosis by the Papanicolaou method is being obtained by rectal lavage. It is a time consuming procedure however and yields relatively few positive results.

The diagnosis of carcinoma should never be based on the results of digital and visual examination alone. Biopsy is essential to differentiate other ulcerating tumors of the lower bowel. These include tuberculoma, amebic granuloma, nonspecific inflammatory granuloma, actinomycotic granuloma and even gumma.

MULTIPLE PRIMARY CARCINOMAS

The concurrent presence of two or even more primary carcinomas in the colon or rectum is not rare, having been reported to occur in 3.7 to 7.5 per cent of cases of large bowel malignant disease (22). Colcock (44) reported that 4 per cent of all patients with carcinoma of the colon or rectum at the Lahey Clinic have a second primary lesion in some segment of the bowel. Berson and Berger (23) stated: "Unquestionably many poor results [in the treatment of carcinoma of the large bowel] can be attributed to the fact that a second lesion was missed. The surgeon should without fail palpate the entire large bowel carefully and routinely in a search for a second primary lesion before proceeding with the definitive treatment of the known and established carcinoma."

TREATMENT

Dukes quotes an apt French proverb: "It is the first step that counts!" This is particularly true of the surgical treatment of colorectal carcinoma, for only at the first operation is it possible to eradicate the primary lesion as well as the metastases. Although this fundamental premise is universally acknowledged, there is no unanimity of opinion on how best to accomplish this. The question

or at autopsy (Fig 87) It is hoped that these newer meticulous, thorough dissections will result in increased salvage of patients afflicted with cancer of the lower rectum

The management of carcinoma of the upper third of the rectum and rectosigmoid area is still a controversial subject It has been well established that in carcinomas of the upper rectum or rectosigmoid the direction of the lymphatic flow is upward and that only rarely, if the upward path of spread has been completely blocked is there lateral and distal spread These important pathologic studies

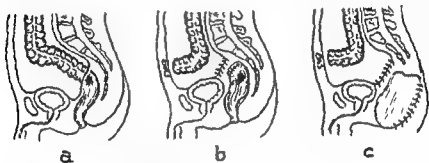


FIG 86—Abdominoperineal resection *a* preoperative state *b* end of abdominal phase with permanent colostomy and peritoneum closed *c* end of perineal phase after resection of tumor rectum and anus

have led to a decided revival of efforts to perform adequate resection of high rectal and rectosigmoidal growths and their lymphatic metastatic routes and then to restore continuity of the bowel thereby preserving anal continence By such technics permanent colostomy with its many physical and psychologic burdens can be avoided In general, these operations aim to preserve sphincter function by (1) preserving only the external sphincter muscle and anus with the sigmoid pulled through and establishing a sigmoidal anal anastomosis (Bibcock Bacon), or (2) intussuscepting the lower rectum pulling through the sigmoid effecting the anastomosis and restoring the intussuscepted anastomosed rectum in o the pelvis (Maunsell Weir, Goligher Black) In the early half of the past decade, inspired by the excellent results of anterior seg

mental resection reported by Dixon and by Wangenstein surgeons investigated the possibility of extending this technic to treating growths as low as the midportion of the rectum. It was soon apparent that when used in lesions below 16 cm (6 in.) from the anal

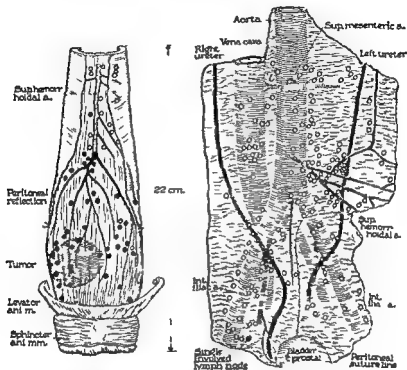


FIG 87—Diagrams of operative specimen (left) showing tumor and involved nodes and autopsy specimen (right) showing solitary involved node in pelvis (From R. L. Gilchrist *Ann Surg* 111 630 1940)

verge recurrences at the line of suture and in the pelvis occurred early and with inordinate frequency (31 32)

Despite the great advantage of preserving anal function the trend to sphincter saving operations in rectal cancer is waning rapidly. However for high rectal lesions Babcock and Bacon continue

the use of abdominoperineal proctosigmoidectomy with preservation of the sphincters. Their results reflect the skill and thoroughness with which they perform this operation. Bacon *et al* (27) reported a 10 year survival rate of 47 per cent, the five year survival rate was 58.4 per cent with a resectability rate of 93 per cent. Despite these praiseworthy results the Babcock-Bacon operation has not become generally popular. The reason for this was summarized by Lahey (33):

It is well known that unless rectal mucosa is preserved for a short distance above the sphincters the function of the rectum will not be preserved. When one realizes as has been frequently stated how delicate and discriminatory rectal mucosa is in permitting the patient to distinguish safely between the desire to expel fecal matter and the desire to expel gas, it can be realized how delicate this sensory apparatus is. If a sphincter preserving operation will hold solids but not liquids, it has little if anything to offer over a well-established colostomy which will do the same.

The control of defecation after the Babcock-Bacon operation depends largely on the attainment of a formed stool mass by diet and other measures, and on the amount of voluntary control remaining in the external sphincter muscles. The studies of Gaston (35), of Goligher and Hughes (36, 37), and of others have shown that retention of the lower rectal mucosa, musculature and nerve supply is essential for satisfactory control. When the distal rectal segment is distended by fecal matter or experimentally, the external sphincter is forewarned of imminent defecation and it contracts. This essential segment is sacrificed in the Babcock-Bacon operation.

For high rectal and rectosigmoidal lesions it is well established that segmental resection and primary anastomosis is the procedure of choice when criteria for restorative resection are satisfied (34). In such cases after operation the function of defecation remains unimpaired because the lower half of the rectum, the sphincters and their nerve supply remain intact. In certain high rectal carcinomas it is found that after resection of the lesion a low rectal stump in a narrow pelvis remains. It is difficult or impossible in such cases to perform a satisfactory and safe primary anastomosis.

In such cases an abdominoanal excision of the Maunsell Weir type is performed. In this operation the abdominal portion of the procedure is the same as for anterior resection. Stay sutures are attached to the rectal stump which is invaginated through the manually dilated anus. The sigmoid is then drawn through the anus and anastomosed to the rectal stump following which the rectum is returned to the pelvis. The peritoneum is then closed above the anastomosis. The anus and about 6 cm. of rectum thus remain intact. In many such cases perfect continence for even liquid feces and gas has resulted (37-38).

In summary, the consensus of contemporary surgeons unequivocally favors abdominoperineal excision with extensive lateral and proximal lymph node dissections for carcinoma of the lower half of the rectum and anus. For high rectal and rectosigmoidal lesions opinion is divided with the majority favoring abdominoperineal resection as the safest procedure. However, when pathologic and physical conditions are favorable, some form of restorative resection may be utilized so that anal continence may be maintained.

In certain specific conditions such as rectal cancer in the extremely senile, in patients with diabetes, nephritis, advanced heart disease, and in the debilitated individual—none of whom could withstand mutilating surgery—the choice must often be made between a radical operation and the life of the patient. For these patients a Hartmann operation is indicated. After the lesion is removed by the abdominal route, the end of the severed rectal stump is inverted and replaced in the pelvis, and an abdominal colostomy is then fashioned. A palliative Hartmann operation is also a reasonable operation in patients with rectal cancer and extensive liver, peritoneal, or lymphatic metastases. In such individuals the perineal phase of the radical operation would serve no useful purpose. For high rectal or rectosigmoidal growths with extensive visceral metastasis, a palliative segmental resection and anastomosis, when possible, will remove the ulcerating toxic lesion and save the patient from colostomy for his few remaining months. Occasionally a patient who is physically handicapped and cannot take care of a

colostomy is seen. This group comprises the blind, the hemiplegics, and those with paralysis agitans or other degenerative diseases. In these patients it is often expedient to perform a restorative resection instead of an abdominoperineal resection, the indicated operation.

Although the scope of operations for lower bowel cancer has been greatly extended, even to complete pelvic exenterations in some instances the disease must be considered inoperable even now. Lahey (39), in answering the question "When is cancer of the colon and rectum inoperable?" said:

One of the things which we have had surprisingly demonstrated to us beyond doubt is the fact that not only is the removal of the malignant lesion in the rectum or the colon justified when there are moderate metastases in the liver because of the fact that life is prolonged and made more comfortable but there is now quite universal agreement on the part of surgeons experienced with the radical removal of malignant lesions of the colon and rectum that in such instances with moderate metastasis in the liver there appears in certain cases to be a retardation effect upon the metastases in the liver by the removal of the primary lesion.

In cancers of the rectum which can be demonstrated by cystoscopy to have invaded the base of the bladder operation is not worth while. In cancers of the rectum which are firmly fixed particularly along the lateral ligament of the rectum and out on to the lateral walls of the pelvis it is likewise not worth while.

RESULTS OF TREATMENT—It has been stated that cure in malignant disease depends directly on complete elimination of the primary tumor and its metastatic channels. Follow up results of treatment of colorectal cancer corroborates this. Fortunately there are a number of long term follow up reports from several outstanding surgical clinics (40-44). Although there was no uniformity of procedure in obtaining and presenting the data significant facts emerge which provide an over all picture of the situation.

The material analyzed reflects the problem as it existed a decade or more ago at a time when our present supplements to surgery were not yet available. These newer surgical aids have reduced postoperative mortality from all causes despite an increased resecta-

bility rate. A review of these comprehensive reports discloses several significant facts that can be summarized.

1. Due to improvements in preoperative preparation, postoperative care, anesthesia, and surgical technique, the operability rate for definitive surgery has risen from about 60 per cent to approximately 85 per cent.

2. The postoperative mortality rate has dropped from about 25 per cent to about 6 per cent.

3. There has been little change in long-term survivals, with perhaps poorer figures in recent cases. This apparent paradox is perhaps explained by the fact that few cases today are considered inoperable, and even patients with advanced lesions are given the hope of definitive resection rather than the palliative colostomy of former years. Grinnell (40) rightly states: "We should not be deterred from continuing to do radical resections on tumors whose curability is undetermined."

4. High salvage rates are possible in the Dukes grade A and even grade B tumors. It follows that early diagnosis is the *sine qua non* for cure. The palpating finger, the sigmoidoscope, and increased and early use of the barium enema in all cases, even suggestive of lower bowel disease, will make possible the early diagnosis of cancer, and even better, will expose the precancerous polyp.

RECURRENT CANCER—Early recurrence of cancer at the suture line is common in segmental resection (31). Goligher *et al.* (45) suggested that during manipulation of the tumor at operation, carcinoma cells are broken off and pass down to lie free in the lumen of the rectum. During subsequent suture, these viable cells are implanted into the suture line or onto an eroded portion of the mucosa (Fig. 88). The case reports and the illustrations of the specimens tend to support this theoretical explanation of the mechanism of this type of local recurrence.

On the assumption that recurrences in many cases must be due to implantation of carcinoma cells on a raw surface, surgeons at St. Mark's Hospital, London, are now routinely washing out the rectum before proceeding with the anastomosis (45). Perhaps this

important contribution to the technic of cancer surgery of the large bowel will help to solve this phase of the problem of local recurrence in sphincter saving operations. In the surgery of cancer Wangenstein has practiced and advocated a second look by exploratory operation several months after the primary operation in all cases showing node metastases. He believes that a fair percentage

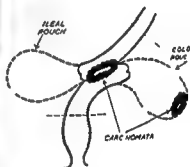


FIG 88—Resected specimen of ileosigmoid anastomosis after subtotal colectomy for carcinomas of transverse and sigmoid portions of colon. Carcinoma recurred three years later at suture line (A) and in blind colon pouch (B) (From J. C. Golligher *et al* *Brit J Surg* 39:199, 1951)

of early recurrences may be found and excised at the second look, thereby increasing the overall salvage rate for colorectal cancer. These hopeful advances in our attack on cancer will be evaluated in time.

TERMINAL CANCER—Care of the hopeless and suffering patient with recurrent or metastatic and progressive cancer of the colon and rectum is a difficult problem. At this stage of the disease the patient usually is returned home to the care of the family physician, whose unhappy task it is to ease the patient and the family through the relentless terminal phase (49). Good nursing care will

do much to relieve discomfort. Cachexia, weakness, and anorexia may be temporarily benefited by a daily intramuscular injection of Teroplerin (sodium pteroyl triglutamate). When effective it improves the sense of well being, increases appetite, and occasionally reduces pain.

Pain must be controlled with minimal doses of drugs or combinations of drugs such as aspirin and a barbiturate. When narcotics become necessary, Dilaudid, Demerol, or Methadone is generally less toxic than morphine for continued use. Slow intravenous infusions of 5 per cent alcohol and 5 per cent dextrose in 1 000 cc of water, alternating every other day with intravenous infusions of procaine 1 Gm in 1 000 cc of saline, will give comfort for varying lengths of time. Patients with metastatic disease involving the floor or walls of the pelvis suffer constant agonizing pain which can be controlled only with toxic doses of narcotics. In such terminal bed-ridden cases, a valuable measure which has resulted repeatedly in considerable or even complete relief of pain is sacral nerve block by intrathecal injection of alcohol. This procedure can be performed in the home. The patient is placed in the inverted prone position; dural puncture is performed by the lateral sacral approach (*see* Chapter 3) and 1 cc of absolute alcohol in a tuberculin syringe is slowly injected through the spinal needle. Because of its low specific gravity, the alcohol floats upward to localize around the sacral nerve roots in the cauda equina. The sacral plexus is distributed to a large portion of the pelvis and perineum and controls their sensory nerves. As these are poorly myelinated, the alcohol readily penetrates the sacral nerve roots to produce a toxic degeneration in the nerve. The patient is maintained in the prone position for an hour to insure fixation of the alcohol. A neurogenic bladder is an expected sequel. This is treated by means of an indwelling catheter and daily irrigations with boric acid solution. A neurogenic bladder in a terminal cancer case is little enough to pay for the invariable relief from intolerable pain. In the occasional case in which pain relief is minimal, the intrathecal injection of an additional 1 cc of alcohol is repeated after three days.

COLOSTOMY

An estimated two thirds of patients with rectal cancer necessarily require abdominoperineal resection and permanent sigmoidal colostomy. A well fashioned and properly functioning colostomy is entirely compatible with the patient's comfort as well as with his continued social and economic well being. The dread of colostomy stems largely from experiences with late terminal colostomies for inoperable obstructive lesions; in these cases the hardship and suffering are indeed agonizing. With better and earlier diagnosis affording increased rate of resectability, it seems likely that fewer palliative colostomies will be necessary.

Temporary colostomies are frequently performed in obstructive resections, in staged operations for congenital anomalies in diverticulitis, and in other conditions. These cases present few problems.

To be easily accessible a sigmoidal colostomy should be placed in the left lower quadrant about midway between the umbilicus and the iliac crest. It should be fashioned so that such complications as retraction, stenosis and prolapse do not occur.

The permanent routine of managing the colostomy is instituted while the patient is still in the hospital. Of great importance is a highly optimistic attitude on the part of the surgeon, the attending physician, and the patient's family, with stress on the fact that the patient's difficulty has been effectively eliminated and all that now remains is for him to adjust his excretory function to a new outlet (46).

The satisfactory functioning of a colostomy depends on many factors. Of prime importance is the response of the colon to the patient's diet; its reaction to the patient's conflicting emotional states following his formidable abdominoperineal resection; and finally the intelligence and determination with which the patient follows the colostomy routine. There is no patient for whom a standard uniform technic of colostomy management can be outlined. The general measures presented here must be adapted to the problems of the individual patient.

DIET—At the onset of colostomy training a constipating diet is best, so that the colostomy will function only on irrigation. By the avoidance of spillage in this early postoperative phase, the patient's confidence in his ability to control the abdominal orifice voluntarily is bolstered. The diet should be high in proteins, moderate in carbohydrates, and low in fats. Excess fats are laxative. Fluids should be limited: tea and cocoa because of their tannic acid content make their use preferable to coffee which acts as a laxative in many individuals. Vegetables high in cellulose are avoided in order to decrease fecal bulk. Deodorized tincture of opium in graduated dosage of 5 to 10 drops is useful in decreasing colonic activity. The patient's food idiosyncrasies and allergies must be taken into account in working out a diet. A simple food for example milk will constipate one individual and cause diarrhea in another. Offending food items are easily discovered by keeping a chart of all ingested foods over a period of time just as is necessary for the discovery of allergic foods. As time progresses and regulation of the function of the colostomy is improved the diet gradually becomes unrestricted.

IRRIGATION—The patient is taught to irrigate the colon while in hospital. An irrigation every other day with 1 to 2 qt of water fractionally or at one time depending on the irritability of the colon is usual in the early phase of colostomy training. The irrigating tube should consist of a colon tube of 30 F diameter with a blunt end. It is introduced 5 to 6 in into the colon and warm tap water is run in. The irrigating can or enema bag is raised no higher than 2 ft above the orifice. The return flow is caught in a curved emesis basin. After a time a colostomy apparatus such as that designed by Binkley (47) and used at the Memorial Hospital New York may be utilized. The purpose of the irrigation is to empty the colon completely so that colonic contents do not issue at an inopportune time. The time required for complete irrigation depends on the rate of return flow whether it returns quickly and in large quantity or slowly and in small dejections. The average time at first amounts to somewhat over an hour. This period can usually be reduced in the course of time.

Constipation can occur and is characterized by extremely dry scybalous stools. Diarrhea also may occur. These variations of function are managed just as one would manage these symptoms in an individual with an intact excretory tract. The passage of flatus is annoying and difficult to control.

The use of colostomy bags is to be discouraged. Petrolatum gauze placed over the colostomy and covered by several layers of gauze or absorbent cotton makes a satisfactory dressing. An elastic girdle for women and an athletic supporter with a wide waist band for men holds the dressing in place.

The individual's adaptation to life with a colostomy is actually not nearly as rosy for all individuals as is pictured by some surgeons. Studies by Dukes, by McLanahan and Gilmore, by Ewing and by Sutherland *et al* indicate that a large percentage of colostomy patients suffer significant impairment of the general functions of active economic and social life. Sexual impotence is a frequent sequel of abdominoperineal resection. It must be stressed that this withdrawal from sexual life may be caused largely by social and psychologic factors. Sutherland *et al* (48) studied 57 patients who had remained free from disease five years or more after abdominoperineal resection for rectal cancer. Their detailed findings with regard to compulsive irrigations, diet, spillage, sexual function, work, function, social participation and psychologic reactions to the surgical experience of the operation contribute greatly to the evaluation of the problem of colostomy. Their report deserves serious study.

LYMPHOSARCOMA OF COLON AND RECTUM

Hayes *et al* (50) reviewed the literature and collected 341 cases of lymphosarcoma of the colon and rectum. Usher and Dixon (51) reported 10 lymphosarcomas of the rectum in 50 cases involving the small and large intestines. In one case personally observed, and later reported by Winkelstein, the rectal mucosa was markedly thickened and indurated, presenting a peculiar cobblestone configuration. The diagnosis is rarely made in an early stage because there are no symptoms at this time. Patients respond well for a time.

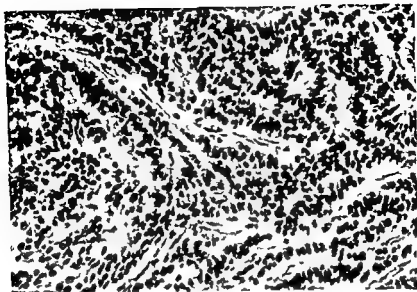


FIG 89 (*above*) —Photomicrograph of small carcinoid of rectum showing ribbons and fasciculi (Figs 89 and 90 from R. C Horn Jr Cancer 2 819 1949)
 FIG 90 (*below*) —Extensive hepatic metastases from same rectal carcinoid

to deep x ray treatment, but eventually die of extensions of the disease

CARCINOID TUMORS

These tumors are found in the ileum and appendix but rarely in the colon and rectum. However, stimulated by the work of Ashworth and Wallace (52) and of Stout (53), colon and rectal carcinoids were intensively studied and reported by Horn (54), Tavener *et al* (55) Rosser (56), and Foreman (57)

These tumors originate in the chromargentaffin cells of Kulitzky which are situated in the crypts of Lieberkuhn. Because they stain deeply with silver salts Masson termed them argentaffin or neurocrine tumors. They proliferate in the submucosa and as a rule are benign, rarely penetrating the serosa or mucosa.

The histologic picture of most rectal carcinoids is of small or moderate sized epithelial cells which grow in cords or ribbons interweaving to form carelessly coiled festoons (Fig 89). Carcinoids of the colon and especially of the rectum may be invasive and exhibit highly malignant metastatic tendencies (Fig 90). A review of the literature to 1953 revealed that a total of 85 carcinoids involving the rectum have been reported of which 10 (11.7 per cent) were malignant as indicated by local and visceral metastases.

Carcinoids of the rectum usually appear as small submucosal freely movable tumors of yellowish hue which average 1 cm in diameter. Those that have been shown to be malignant were somewhat larger usually ulcerated and grossly could not be differentiated from carcinoma. Local excision suffices for small movable submucosal tumors. If the tumor is ulcerated large and fixed it must be presumed to be malignant and radical abdominoperineal excision is essential.

REFERENCES

- 1 American Cancer Society Statistical Research Section: Cancer Deaths by Site (New York 1949)
- 2 Ottenheimer E J: Cancer of the rectum. New England J Med 23: 1 1947
- 3 Jackman R J, Niebling H A and Waugh J M: Carcinoma of the large intestine. JAMA 134 1287 1947

- 4 Granet E. Diagnosis of early cancer of the colon and rectum *Am J Digest Dis* 17 95 1950
- 4a. Maes U. and Essig I. M. Some reflections on surgical principles in treating cancer of the colon and rectum *Ann Surg* 130 1008 1949
- 5 Hodges T. J. and MacMillan H. C. Important clinical dividends from x ray examination of the colon *JAMA* 147 1191 1951
- 6 McQuarrie H. B. and Buie L. A. Epithelioma of the anus *Postgrad Med* 7 402 1950
- 7 Binkley G. E. Epidermoid carcinoma of the anus and rectum *Am J Surg* 79 90 1950
- 8 Sauer I. and Bacon H. E. A new approach for the excision of carcinoma of the lower portion of the rectum and anal canal *Surg. Gynec & Obst* 95 229 1952
- 9 Roux Berger J. L. and Ennuyer A. Carcinoma of the anal canal *Am J Roentgenol* 60 807 1948
- 10 Friedman M. Personal communication 1953
- 11 Raven R. W. Anorectal malignant melanoma *Am J Surg* 79 85 1950
- 12 Braastad F. W. Dockerty M. H. and Dixon C. F. Melano-epithelioma of the anus and rectum *Surgery* 25 82 1949
- 13 LeBlanc L. J. Buie L. A. and Dockerty M. B. Squamous-cell epithelioma of the rectum *Ann Surg* 131 392 1950
- 13a. Jackman R. J. and Mayo C. W. The adenoma-carcinoma sequence in cancer of the colon *Surg. Gynec & Obst* 93 327 1951
- 14 Broders A. C. Carcinoma grading and practical application *Arch Path* 2 376 1926
- 15 Broders A. C. Sr. Phillips C. and Stinson J. C. Neoplasms of the large bowel *S Clin. North America* 32 1511 1952
- 16 Dukes C. E. The surgical pathology of rectal cancer *Am J Surg* 79 66 1950
- 17 Grinnell R. S. The spread of carcinoma of the colon and rectum *Cancer* 3 641 1950
- 18 Connell J. F. and Rottino A. Retrograde spread of carcinoma in the rectum and rectosigmoid *Arch Surg* 59 807 1949
- 18a. Quer E. A. Dahlin D. C. and Mayo C. W. Retrograde intramural spread of carcinoma of the rectum and rectosigmoid *Surg. Gynec & Obst* 96 24 1953
- 19 Sunderland D. A. Significance of vein invasion by cancer of the rectum and sigmoid *Cancer* 2 429 1949
- 19a. Gilchrist R. K. and David V. C. Lymphatic spread of carcinoma of rectum *Ann Surg* 108 621 1938
- 20 Gilchrist R. K. Fundamental factors governing lymphatic spread of carcinoma *Ann Surg* 111 630 1940
- 21 Gilchrist R. K. and David B. C. Pathological factors influencing five year survival in radical resection of the large bowel and rectum for carcinoma *Ann Surg* 126 421 1947
- 22 Brindley G. V. and Rice J. S. Multiple primary malignancies of the large intestine *S Clin North America* 32 1499 1952

- 23 Berson H L and Berger L Multiple carcinomas of the large intestine Surg Gynec & Obst 80 75 1945
- 24 Gladstone S A Sponge biopsy A new method in the diagnosis of cancer Cancer 2 604 1949
- 25 Graham A S Current trends in surgery of the distal colon and rectum for cancer Ann Surg 127 1072 1948
- 26 Appleby L H Treatment of advanced cancer of the rectum Proc Roy Soc Med 43 1071 1950
- 27 Bacon H E McElwain J W and Trumps H D Surgical management of large bowel lesions Bull New York Acad Med 29 34 1953
- 28 Deddish M R Treatment of advanced cancer of the rectum Proc Roy Soc Med 43 1075 1950
- 29 Ault G W Castro A F and Smith R S A clinical study of ligation of the inferior mesenteric artery in left colon dissection Surg Gynec & Obst 94 223 1952
- 30 State D Combined abdominoperineal excision of the rectum Surgery 30 349 1951
- 31 Garlock J H and Ginzberg L Appraisal of anterior resection for carcinoma of the rectum and rectosigmoid Surg Gynec & Obst 9 525 1950
- 32 Judd E S Jr and Bellegie N J Carcinoma of the rectosigmoid and upper part of rectum A M A Arch Surg 64 697 1952
- 33 Lahey F H Preservation of the sphincter operations in carcinoma of the rectum Lahey Clin Bull 7 162 1951
- 34 Morgan C N and Lloyd Davies O V Restorative resection of the rectum Proc Roy Soc Med 43 701 1950
- 35 Gaston E A The physiology of fecal continence Surg Gynec & Obst 87 280 669 1948
- 36 Goligher J C and Hughes E S R Sensibility of the rectum and colon Lancet 1 543 1951
- 37 Goligher J C The functional results after sphincter saving resections of the rectum Ann Roy Coll Surgeons England 8 421 1951
- 38 Black H M Combined abdominoendorectal resection A M A Arch Surg 65 406 1952
- 39 Lahey F H When is cancer of the rectum and colon inoperable? Lahey Clin Bull 6 194 1950
- 40 Grinnell R S Results in treatment of carcinoma of the colon and rectum Surg Gynec & Obst 96 31 1953
- 41 Garlock J H and Klein S H Carcinoma of the colon and rectum A ten year study Arch Surg 59 1269 1949
- 42 Binkley G E and Stearns M W Jr Ten year surgical results of rectal cancer Surg Gynec & Obst 93 428 1951
- 43 Collier F A, *et al* Cancer of the rectum A study of long term survival Ann Surg 135 841 1952
- 44 Colcock B P In discussion on Collier *et al* (43)
- 45 Goligher J C Dukes C E and Bussey H J R Local recurrences after sphincter saving excisions of carcinoma of the rectum Brit J Surg 39 199 1951

- 46 Lyons A S Care of the colostomy J Mt Sinai Hosp 18 39 1951
- 47 Binkley G E Construction and care of the abdominal colostomy Am J Surg 83 807 1952
- 48 Sutherland A M *et al* The psychological impact of cancer adaptation to the dry colostomy Cancer 5 857 1952
- 49 Treves N Problems in the postoperative care of cancer patients New York State J Med 44 2248 1954
- 50 Hayes H T *et al* Lymphoid tumors of colon and rectum Tr Am Proct Soc 40 214 1939
- 51 Usher F C and Dixon C F Lymphosarcoma of the intestines Gastroenterology 1 160 1943
- 52 Ashworth C T and Wallace S A Unusual locations of carcinoid tumors Arch Path 32 272 1941
- 53 Stout A P Carcinoid tumors of the rectum Am J Path 18 993 1942
- 54 Horn R. C. Jr Carcinoid tumors of the colon and rectum Cancer 2 819 1949
- 55 Tavenner M C Bacon H E. and Peale A R Carcinoid tumors of the rectum J Internat Coll Surgeons 16 265 1951
- 56 Rosser C Carcinoid (neurocrine) tumors of the rectum Surg Gynec & Obst 93 486 1951
- 57 Foreman R. C. Carcinoid tumors Ann Surg. 136 838 1952

Ulcerative Colitis

CHRONIC ULCERATIVE COLITIS

THE SPECIFIC CAUSE of chronic ulcerative colitis is unknown. The clinical, pathologic and psychologic aspects are well documented in most textbooks of clinical medicine and need not be considered here at length. It is the complicated problem of the management of the patient with colitis that will be discussed here in detail.

CLINICAL FORMS—Nonspecific ulcerative colitis manifests itself in several clinical forms. In the order of relative frequency, the disease occurs as (1) localized proctosigmoiditis, (2) generalized and left sided colitis, (3) segmental colitis, (4) right sided colitis and (5) ileocolitis. The mild type with few symptoms is most often seen; the severe recurrent debilitating form of the disease is less frequent; and the fulminating toxic, and often rapidly fatal form fortunately is rare.

Symptoms in the mild forms are limited to frequent defecations of semisolid mucoid feces which occasionally are sanguineous. Nutrition is well maintained, abdominal pain is inconstant and there is no toxemia. If the disease involves the sigmoid and rectum sigmoidoscopy reveals the mucosa to be covered by a sanguineous exudate containing flecks of mucus. The mucosa appears lusterless, edematous, granular, injected and so friable that it bleeds at the slightest touch. There is no ulceration in mild cases early in the disease.

Barium enema reveals no abnormal changes in the appearance of the colon except perhaps increased spasm

The patient with long standing ulcerative colitis rarely passes formed stools. Characteristically, his daily bowel dejections consist of five to 15 liquid stools which contain varying quantities of bloody mucus. Severe tenesmus is constantly present to which are added generalized abdominal cramps, anorexia, loss of weight, secondary anemia and multiple manifestations of avitaminosis.

The x-ray picture of chronic ulcerative colitis depends on the chronicity of the disease. The postevacuation film and the aerogram often reveal distortion of the delicate filigreed pattern formed by normal colonic rugae. In chronic colitis the pattern is fuzzy, indistinct or absent in the diseased areas. These changes reflect distortion caused by mucosal swelling or destruction. The aerogram reveals a thickened bowel wall and may disclose inflammatory pseudopolyps. With prolonged chronicity fibrosis and thickening of the bowel wall take place resulting eventually in contraction of the lumen, shortening of the bowel and loss of mobility—the characteristic lead pipe deformity of chronic ulcerative colitis. Exacerbations in this chronic and recurring disease are common during which severe toxemia, high fever, rapid depletion of electrolytes, exsanguinating hemorrhages and marked prostration are characteristic.

PATHOLOGY—Material for pathologic study has been obtained in intractable cases after colectomy or at autopsy. In these specimens a tremendous thickening of the submucosa, hypertrophy of the muscle coat and a general shortening of the whole length of the large bowel have been found. Superficial ulceration involving most of the mucous membrane is always present and disseminated polypoid lesions may be

Meyer *et al.* (1) found a pronounced increase in the lysozyme titer of the colonic secretions in cases of ulcerative colitis. This enzyme is found in various body secretions including tears, nasal secretion, saliva and intestinal secretions and also in egg white. Its action is bacteriolytic and mucolytic. The concentration of lysozyme

Proctology

in the stools of ulcerative colitis patients exceeds that of normal stools by an average of 27 times Meyer *et al* also showed that the mucolytic effect of lysozyme on intestinal surface mucus deprives the mucosa of its normal protective covering and exposes its cellular structure to the erosive action of intestinal proteolytic enzymes and secondary bacterial infection. The lysozyme titer is a useful index of progress in cases of ulcerative colitis, the lysozyme concentration falling almost to zero with progressive clinical remission.

Levine *et al* (2) noted virtual absence of homogeneous ground substance in the basement membrane of epithelial cells in biopsy specimens from patients with ulcerative colitis. They believe that as changes in the basement membrane occur the epithelium sloughs away from the submucosal connective tissue permitting secondary bacterial invasion to supervene. This concept of the pathogenesis of chronic ulcerative colitis is intriguing but so far lacks confirmation. Storsteen, Kernohan and Borgen (3a) studied the myenteric plexuses (Auerbach's) in colons from cases of ulcerative colitis obtained at autopsy and at operation. The number of ganglion cells (see Fig 25 N) in the diseased colons was increased approximately threefold over the ganglion cell count in the myenteric plexuses of normal colons. Most morphologists hold that the ganglion cells of the myenteric plexus belong to the parasympathetic system which regulates peristalsis and secretion. Future investigations may show that the hyperactive motor and secretory phases of ulcerative colitis may be related in some way to the marked increase in the number of ganglion cells.

PSYCHOSOMATIC ASPECTS—It has been conclusively demonstrated that emotional disturbances in the life situation of patients adversely affects the course of the disease. The personality make up of the patient has been described as that of an emotionally immature parentally attached dependent individual lacking fulfillment of his life's endeavors (3).

Crohn and Yarnis (4), Portis (5) and Almy (6) discuss clearly and at length the various phases of the psychologic problems presented by the patient with ulcerative colitis. They stress the

need for a warm sympathetic, and understanding relationship between physician and patient throughout the period of medical management which is essential to bring the patient into the stage of remission. Only then should an attempt be made to probe further into the personality difficulties of the subject. However, this must be done skilfully and must be kept on a rather superficial level.

The effect of the autonomic nervous system on the distal colon is twofold. The sacral parasympathetics when stimulated increase smooth muscle contraction or spasm as well as arteriolar vasospasm resulting in edema, venous stasis and finally in mucosal friability. Atropine or related anticholinergic drugs help to allay spasm. Shafiroff and Hinton (7) performed pelvic autonomic neurectomy on animals and on patients with ulcerative colitis and had encouraging results.

Vagotomy has been tried in treatment of colitis and regional ileitis. Eddy (8) reporting results in 42 patients followed up to 19 months after treatment stated that intestinal motility and spasm generally decreased resulting in favorable clinical response in many patients. He believes however that vagotomy should not be used in chronic cases of long standing, i.e. five years or more.

COMPLICATIONS—Local lesions complicate long standing chronic ulcerative colitis. In 2 000 cases reviewed by Sloan *et al* (9) the following lesions were found:

Lesion	No. of Cases
Polyps	19
Stricture	11
Anorectal infection (abscess fistulas etc.)	8
Cancer	5
Perforation	2

On the whole statistics in the literature on the incidence of complications agree with the foregoing figures. A review of the histories of 120 patients with severe ulcerative colitis on the ward service of Mount Sinai Hospital, New York, revealed a higher percentage of local lesions (10).

in the stools of ulcerative colitis patients exceeds that of normal stools by an average of 27 times Meyer *et al* also showed that the mucolytic effect of lysozyme on intestinal surface mucus deprives the mucosa of its normal protective covering and exposes its cellular structure to the erosive action of intestinal proteolytic enzymes and secondary bacterial infection. The lysozyme titer is a useful index of progress in cases of ulcerative colitis the lysozyme concentration falling almost to zero with progressive clinical remission.

Levine *et al* (2) noted virtual absence of homogeneous ground substance in the basement membrane of epithelial cells in biopsy specimens from patients with ulcerative colitis. They believe that changes in the basement membrane occur the epithelium sloughs away from the submucosal connective tissue permitting secondary bacterial invasion to supervene. This concept of the pathogenesis of chronic ulcerative colitis is intriguing but so far lacks confirmation.

Storsteen, Kernohan and Barger (3a) studied the myenteric plexuses (Auerbach's) in colons from cases of ulcerative colitis obtained at autopsy and at operation. The number of ganglion cells (see Fig 25 N) in the diseased colons was increased approximately threefold over the ganglion cell count in the myenteric plexuses of normal colons. Most morphologists hold that the ganglion cells of the myenteric plexus belong to the parasympathetic system which regulates peristalsis and secretion. Future investigations may show that the hyperactive motor and secretory phases of ulcerative colitis may be related in some way to the marked increase in the number of ganglion cells.

PSYCHOSOMATIC ASPECTS—It has been conclusively demonstrated that emotional disturbances in the life situation of patients adversely affects the course of the disease. The personality make up of the patient has been described as that of an emotionally immature parentally attached dependent individual lacking fulfillment of his life's endeavors (3).

Crohn and Yarnis (4) Portis (5) and Almy (6) discuss clearly and at length the various phases of the psychologic problems presented by the patient with ulcerative colitis. They stress the

need for a warm sympathetic and understanding relationship between physician and patient throughout the period of medical management which is essential to bring the patient into the stage of remission. Only then should an attempt be made to probe further into the personality difficulties of the subject. However this must be done skilfully and must be kept on a rather superficial level.

The effect of the autonomic nervous system on the distal colon is twofold. The sacral parasympathetics when stimulated increase smooth muscle contraction or spasm as well as arteriolar vasospasm resulting in edema, venous stasis and finally in mucosal friability. Atropine or related anticholinergic drugs help to allay spasm. Shafiroff and Hinton (7) performed pelvic autonomic neurectomy on animals and on patients with ulcerative colitis and had encouraging results.

Vagotomy has been tried in treatment of colitis and regional ileitis. Eddy (8), reporting results in 42 patients followed up to 19 months after treatment stated that intestinal motility and spasm generally decreased resulting in favorable clinical response in many patients. He believes however that vagotomy should not be used in chronic cases of long standing i.e. five years or more.

COMPLICATIONS—Local lesions complicate long standing chronic ulcerative colitis. In 2,000 cases reviewed by Sloan *et al* (9) the following lesions were found:

Lesion	No. of Cases
Polyps	19
Stricture	11
Anorectal infection (abscess fistulas etc.)	8
Cancer	5
Perforation	2

On the whole statistics in the literature on the incidence of complications agree with the foregoing figures. A review of the histories of 120 patients with severe ulcerative colitis on the ward service of Mount Sinai Hospital, New York, revealed a higher percentage of local lesions (10).

Lesion	No. of Cases
Anorectal infection (abscess fistulas etc.)	175
Anal ulcer	92
Stricture	58
Perforation	25
Rectovaginal fistula	25

In ulcerative colitis of long standing pseudopolypoid degeneration of the mucosa may occur at the site of healing ulcerations. The polyps usually are sessile, fibrotic and covered with low colonic mucosal epithelium. Orin and Snapper (11) have emphasized the fact that true adenomatous polyps may be present concurrently with inflammatory pseudopolyps. They believe that the malignant changes which occur in the polyposis of ulcerative colitis always arise in the adenomatous polyps, never in the inflammatory pseudopolyps.

The incidence of carcinoma in patients with ulcerative colitis is high. Lyons and Garlock (12), reviewing the subject of this relationship cited Carrell who reported that malignant colonic tumors were present in 7 per cent of all patients who come to surgery and furthermore that among those with the disease for nine or more years carcinoma developed in every third patient. Lyons and Garlock also emphasized the fact that carcinoma develops at an early age in ulcerative colitis that the growths are particularly virulent and because of widespread metastases cause early death. Among 226 patients with ulcerative colitis treated surgically nine carcinomas were removed, an incidence of 4.9 per cent. 25 of the patients had had ulcerative colitis for over 12 years and all nine carcinomas (36 per cent) occurred in this group. The morbid significance of chronic ulcerative colitis is clear from these reports. The importance of periodic sigmoidoscopic and roentgenographic examinations of patients with ulcerative colitis is stressed by all experienced observers.

Among systemic manifestations frequently associated with long standing ulcerative colitis are infectious osteoarthritis erythema nodosum ulcerative trophic skin lesions infectious keratitis and conjunctivitis.

PREGNANCY IN CHRONIC ULCERATIVE COLITIS—The influence of pregnancy on the course of chronic ulcerative colitis has been investigated in a number of clinics. Medicolegal aspects are involved because occasionally pregnancy must be interrupted in a patient with a severe or complicated form of the disease.

Kallet (28) reviewed the literature on this question and concluded from it and from his own observations that most patients proceed to term with little or no toxemia due to the colitis; the effect of the pregnancy on the course of the colitis, however, is not quite so favorable. In 197 gestations in patients with ulcerative colitis the colitis began during pregnancy in 14, was improved by pregnancy in 60, was aggravated during pregnancy in 79, and remained stationary in 44. Kleckner *et al.* (29) at the Mayo Clinic also studied this problem and came to the conclusion that women with ulcerative colitis should not become pregnant unless the disease has been in remission for a long time. This reasonable view reflects the attitude at Mount Sinai Hospital.

MEDICAL MANAGEMENT

As in other debilitating medical diseases of unknown etiology, the treatment of patients with ulcerative colitis is directed toward supporting the patient's nutrition and his metabolic, hematologic, and electrolyte balance. The importance of maintaining and bolstering his psychologic state has been emphasized. Sedation for pain is essential to allow the patient to obtain sufficient rest. Fortunately, about 75 per cent of patients with ulcerative colitis are ambulatory and fall into the general group with mild subjective and objective symptoms. A bland, high protein diet supplemented with multivitamins and essential minerals and necessary sedation are prescribed. Tea, milk, and cocoa are allowed in reasonable quantities but not coffee, which generally is laxative. The frequency of irritating dejections can be diminished and the stools made less excoriating by administering increasing quantities of the hydrophilic colloids with added kaolin or barium sulfate to optimal dosage. The resulting stool has body though soft, is bland, and is hygroscopic.

Proctology

for irritating intestinal secretions Deodorized tincture of opium, in 5 drop doses, is often helpful when taken on arising. The patient should be encouraged to continue his usual occupation and social activities if these are normally pleasant and are not an irritating emotional hazard. He should retire early and spend most of the weekend reclining or in bed.

The prolonged use of thiouracil has been advocated for the treatment of ulcerative colitis because of its cholinesterase activity which tends to inhibit parasympathetic impulses. Encouraging results have been reported following its use in moderately advanced cases of ulcerative colitis (13).

If at all possible, the severely ill patient should be treated in the hospital. The toxicity, constant blood loss, electrolyte imbalance and dehydration as well as the possible systemic complications which may develop require active medical treatment.

A clinical and laboratory appraisal determines the necessary replacement treatment with blood electrolytes, amino acids and vitamins. The serum levels of sodium, potassium and chlorides may be dangerously low and must be quickly raised by parenteral therapy. Sedation is given so that rest is assured. Deodorized tincture of opium 10 minims as often as every three hours may control the diarrhea to some extent. If toxic symptoms are pronounced it may be advisable to discontinue all oral feedings for several days and rely entirely on parenteral alimentation. Multivitamins including B₁₂ are given by injection. Since red cells and hemoglobin are depleted the blood balance must be supported by repeated transfusions if necessary. The possibility that the colitis may be amebic in origin justifies a trial course of emetine. Occasionally there is dramatic cure.

Although not specific for ulcerative colitis, the sulfonamides and broad spectrum antibiotics do limit bacterial infection and toxic absorption from the large areas of denuded colonic mucosa. To circumvent the varying degree of resistance to antibiotics which the ordinary intestinal micro-organisms quickly develop, Crohn and Yarnis (4) have advised the use of phthalylsulfathiazole for a pe-

riod of three weeks followed by chloramphenicol the fourth week

In severe ulcerative colitis uncomplicated by hemorrhage obstruction or polyps management with hyperpyrexia with total parenteral alimentation or with the steroid hormones should be attempted Should one or all of these heroic measures fail ileostomy and colon resection cannot be delayed any longer

Hyperpyrexia induced by intravenous injection of graded doses of typhoid vaccine was used successfully by Wilkinson and Smith (14) Of their 40 patients with ulcerative colitis 68 per cent were greatly improved and 20 per cent moderately improved An initial dose of 3 million killed organisms diluted in 5 cc of saline is injected slowly into a vein If a chill does not follow within two hours the dose of organisms is increased by 50 per cent in a daily intravenous injection until a chill does occur The initial effective dose was found to be 3 to 15 million organisms The optimal dose should produce an effective chill one to three hours after the injection and be followed by a temperature of about 39.5 C (103 F) which remains elevated for about three hours An increase of 50 per cent over the previous dose is made to maintain the temperature at effective levels Treatments are given every other day and continued for a course of 10 injections If treatment is successful it should be continued at two week intervals at the patient's home the dose being approximately one third of the last hospital dose Should the patient react unfavorably i.e. with prostration or exacerbation of the colitis this form of therapy is abandoned

Based on his extensive experience with this disease Paulson (15) evolved a regimen of management for the intractable type of ulcerative colitis which he termed total parenteral alimentation and management For 14 to 21 days or even longer the digestive tract is placed totally at rest by interdicting ingestion by mouth of every thing including water This reduces secretory excretory absorptive and motor activities to a minimum It deflates the intestines and reduces bacterial flora Water electrolytes protein hydrolysate and blood are administered by intravenous injection Vitamins are given intramuscularly Only ice in small quantities is allowed by mouth

Paulson reported that in two thirds of his cases diarrhea was reduced in a few days. Gross blood disappeared from the stools within two weeks. Before feeding is resumed, succinylsulfathiazole is given and continued. Patients who fail to respond to this rigid management in 21 days are considered failures for the method and medically intractable.

Ulcerative colitis seldom starts as a fulminating disease. The fulminating phase which may occur suddenly during the chronic course of the disease is characterized by high fever, tachycardia, pronounced toxemia and prostration, constant diarrhea, abdominal pain and distention. Massive hemorrhage and even perforation occur. The course is unpredictable. Death may ensue rapidly or there may be a sudden unexplained remission.

Although a decided risk in a disease with ulcerative intestinal lesions, a trial with the steroid hormones should be made when all else has failed in the intractable or fulminating cases. The early enthusiasm for treatment with corticotropin and cortisone has been tempered by less favorable experiences following their wider use. These hormones are given in full dosage. Favorable response in previously refractory patients is manifested by signs of remission consisting of decreased number of dejections and diminished fecal blood loss, striking decrease in the lysozyme titer, a sense of well being replacing depression and improved appetite with resulting gain in weight (16). Perhaps the widest indication for the use of the hormones is complicating arthritis. Subjective improvement is not paralleled by improvement in the mucosal ulcerative lesions as observed sigmoidoscopically, the lesions remain unchanged for a prolonged period. This should be expected since the effect of these agents is normally fibrolytic (17). At Mount Sinai Hospital no striking improvement has been noted in the usual case of ulcerative colitis although the effect on systemic symptoms has indeed been favorable, patients gaining weight and having generally an improved sense of well being. It has been our experience in most cases that when this therapy was discontinued or the dosage decreased the symptoms both subjective and objective reverted to their previous

status The great value of the hormones is in preparing patients when surgical treatment of the disease becomes necessary

Many patients with ulcerative colitis continue to deteriorate despite the very best treatment with an unrestricted armamentarium of medication vitamins blood and antibiotics The vital problem of saving the life of such a patient by surgical treatment now arises and must be decided before deterioration progresses to the point where surgery itself is dangerous Resort to heroic measures is justifiable at this time

SURGICAL MANAGEMENT

When the disease has become intractable and irreversible as demonstrated by repeated recurrences and chronic invalidism only an operation will rehabilitate the patient and possibly save his life As in major surgery for other conditions accumulated experience and the support given by antibiotics blood electrolytes and vitamins have materially reduced the former high mortality of ileostomy and colectomy in ulcerative colitis It is generally agreed that surgical removal of the massive focus of infection in the ulcerated colon is necessary when major complications are present (Fig 91) Furthermore the surgery must be done before the patient's metabolic and protein reserve is so depleted that the stress of operation will result in irreversible shock or if the operation itself is weathered in impaired and delayed tissue repair The importance of this may be judged from the fact that 39 per cent of the patients undergoing surgery for ulcerative colitis in one series at the University of Pennsylvania Hospitals had some form of postoperative complication (18)

Lahey (19) gave the following indications for ileostomy and colectomy (1) acute fulminating course (2) medical failure (3) massive hemorrhage (4) obstruction (5) subacute perforations abscess peritonitis intestinal fistula (6) polyposis (7) malignant degeneration (8) joint infections (9) anal incontinence extensive anal fistulas He stated "The only thing I should like to urge ■ do not wait until an acute fulminating case reaches the

stage where no surgeon, no matter how much experience he has had with ulcerative colitis, can do an ileostomy without a high mortality

The percentage of patients with chronic ulcerative colitis who

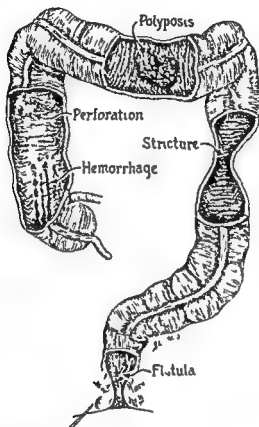


FIG 91 —Indications for ileostomy and colectomy in ulcerative colitis (Figs 91-93 from C. B. Ripstein *et al* Ann Surg. 135:14, 1952)

eventually must be operated on varies widely. At the Lahey Clinic 308 (40 per cent) of 770 patients with ulcerative colitis under treatment required surgical measures. At Roosevelt Hospital, New York, 27 per cent of 500 patients were treated surgically. The latter

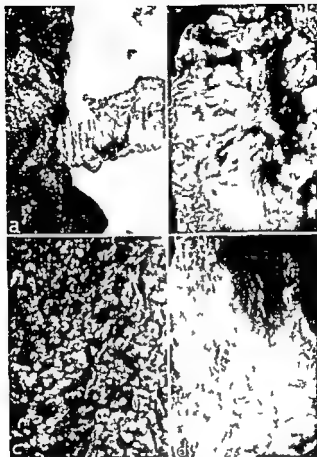


FIG 92—Gross lesions of ulcerative colitis in specimens of excised colons
a acute fulminating colitis with hemorrhage ileum grossly normal *b* acute ulceration with perforation *c* chronic ulcerative colitis with pseudopolyposis *d* chronic colitis with atrophy of mucosa bowel was a fibrous nonfunctioning tube

figure probably approaches more nearly the percentage in most clinics. However, it must be remembered that only patients with the severest forms of ulcerative colitis are seen in large surgical clinics. A fair over all estimate of surgically treated ulcerative colitis is probably about 10 per cent of all types of the disease.

Formerly the surgeon, in a last desperate attempt to save life in fulminating and critical cases had to perform an ileostomy. The purpose of the ileostomy is to remove the colon as a functioning viscus. By thus placing the colon at rest absorption of toxins is minimized and ulcerations tend to heal. The benefit to the patient is often dramatic as manifested by general improvement in symptoms and gain in weight and vigor. The patient so treated does well. However to remain indefinitely a useless, infected and ulcerated colon in which dangerous polyps are very likely to form is now considered not to be good medical practice (Fig 92). Most surgeons feel that only after the removal of the infected ulcerated bowel can the patient be considered cured. Frequently toxemia, hemorrhage and systemic complications persist despite ileostomy and a defunctionalized colon. Colectomy and proctectomy must follow soon after ileostomy in such cases.

Surgeons used to wait six months to years for optimal improvement before proceeding with colectomy. Now Ripstein *et al* (21) perform primary resection of the colon in conjunction with ileostomy. Removal of the diseased bowel immediately eliminates the factors of blood and protein loss as well as of toxic absorption (Fig 93). The problem of postoperative management is simplified and the operative mortality diminishes. Bicon and Trimpf (23) also perform primary colectomy with ileostomy and agree that the over all results are better than with segmental operations.

To the patient with ulcerative colitis faced with imminent surgery the prospect of living with a permanent constantly discharging intestinal stoma is a powerful blow to his already precarious psychologic balance. Ileostomy has its inconveniences, its specific problems in management and its hazards. A forthright but sympathetic explanation of the need for surgery and assurance that

the presence of an ileostomy is compatible with maintenance of social economic, and family relationships will often result in a hopeful, philosophic acceptance by the patient that the ileostomy is preferable to suffering and chronic invalidism. That it is entirely compatible with an active existence is shown by a study of the social and economic status of 104 patients with ileostomy McKirtrick and

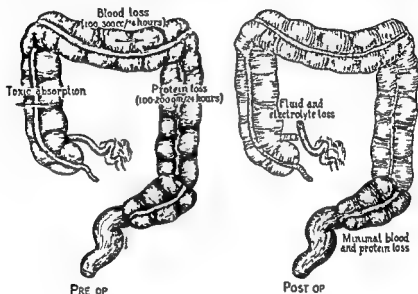


FIG 93—Advantages of colectomy in ulcerative colitis

Moore (24) found that 97 per cent were working as housewives or gainfully employed and that only 33 per cent felt that ileostomy interfered in any way with their social lives. 88 per cent of the patients were in good health. The operation must not be undertaken until the patient and his family know exactly what is contemplated and accept the fact that the ileostomy is likely to be permanent.

The rehabilitation of a patient with an ileostomy can be greatly aided by enlisting the help of well adjusted ileostomy veterans in encouraging the neophyte still beleaguered with doubts and me

chanical problems Lyons (25) has described an Ileostomy Club where veterans of the operation meet on a social basis and, among other activities, discuss freely the problem presented by the ileostomy and their individual adjustment to these problems. He found this means of group therapy valuable.

The care of an ileostomy has been greatly simplified by the use of a flat, form fitting thin rubber bag which fits closely around the

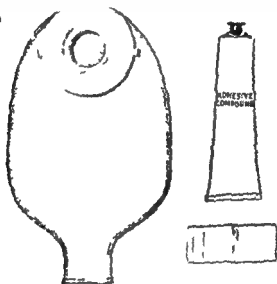


FIG 94—Ileostomy bag (Torbot)

stoma and is sealed to the skin by a special cement. It requires emptying about three times a day. The Koenig Rutzen, the Perma and the Torbot (Fig 94) are satisfactory ileostomy bags.

Although ileostomy and colectomy are essential to success in the surgical treatment of ulcerative colitis, a stump of distal sigmoid and rectum is retained. The final proctectomy is often deliberately delayed in the hope that in an occasional patient with minimal or no rectal involvement it may be possible to restore continuity by a future ileosigmoidostomy. Experience has shown, however, that in

anastomosis is fraught with danger for the disease in the rectal stump often recurs in so virulent a form that it rapidly involves and ascends along the ileum Garlock (27) restored continuity in seven patients in four it was completely successful Cave (27a) and Winslow attempted reanastomosis in six cases, two were successful but in the other four patients ascending ileitis rapidly supervened so that the involved portion of the ileum had to be resected and the ileostomy restored Periodic examination of these defunctionalized rectums after ileostomy reveals that only occasionally does the rectum completely revert to normal appearance In the majority, the mucosa remains edematous injected and friable in many instances contraction of the lumen and eventual stricture in the rectum supervenes

CIRCUMSCRIBED HYPERTROPHIC PROCTITIS

This syndrome was described in 1938 by Fansler and Anderson (30) They considered it an inflammatory infection of the rectum having specific characteristics which distinguish it from ordinary proctitis Usually the first symptom noted by the patient is bleeding with defecation Examination at this stage with the anoscope reveals an injected edematous granular mucosa which is friable and bleeds at the slightest touch The lesion surrounds the sphincteric rectum in a cufflike manner it extends upward for 5 to 8 cm and usually ends abruptly on the distal aspect of the lowest valve of Houston The transition from markedly inflamed edematous mucosa to normal mucosa is sharp and dramatic

At a later stage the marked clinical and pathologic features are more pronounced There is a constant dull ache in the rectum which is often quite severe Pain is present during and after defecation Rectal tenesmus and urgency result in several movements daily often productive only of blood and small amounts of mucopurulent dejections Anal pain is marked so that adequate examination often requires anesthesia In these cases the mucosa is seen to be thrown up into thick hypertrophic folds or ridges corresponding in location to the columns of Morgagni Deep sulci with ulcerated bases which

are present between the folds may extend into or even through the anal canal and are the cause of the characteristic pain. Since its description 15 years ago, I have seen 14 such cases, most of them in early stages of the disease.

While this condition may not be a unique or new type of colitis it does present special problems in treatment. Retention enemas of cod liver oil, sedative ointments and large doses of vitamin C by mouth comprised the treatment in most of my cases, but at no time did I feel that the treatment had a favorable result although remissions and recurrences were frequent. Triple Sulfa Cream instilled through an attached pile pipe, has been tried in some recent cases, over a period of time, both lesion and symptoms seem to have responded favorably.

AMEBIC COLITIS

In the United States, amebiasis is an endemic disease with an incidence of 1 to 40 per cent depending on geographic locality. A careful investigation of a representative group of residents of the Albany N.Y. area who were presumed to be healthy revealed that 13 per cent of those examined were infected with *Endamoeba histolytica* (31). It is assumed that among individuals with previous residence travel or military service in the tropics the incidence of amebiasis is greater.

The symptoms of most patients with amebiasis are mild rarely are they severe enough to cause the patients to seek medical attention. In 34 patients with proved amebiasis Towse (31) found that 15 per cent were symptom free 53 per cent had symptoms of mild colitis 30 per cent had moderately severe symptoms of chronic colitis and one patient (3 per cent) had severe amebic colitis.

The infestation is caused by ingestion of cysts of *E. histolytica*. The motile (vegetative) forms the trophozoites are tissue parasites and live in the submucosa of the intestine as well as in the liver. Cyst formation takes place as soon as the environment becomes unfavorable to the ameba. To cure amebiasis both trophozoites and the cysts must be destroyed. Amebas cause tissue necrosis without

actual suppuration Amebiasis of the rectum takes the form of small indurated areas with necrotic centers 3 to 8 mm in diameter Large necrotic ulcers may be present in the cecum and ascending colon but are rarely present in the rectum The mucosa of the rectum between individual lesions is usually not inflamed The margin of the ulcer is hyperemic and overhangs the ulcer crater giving the latter a punched-out appearance Scrapings taken from the base of the ulcer often contain *E. histolytica* in large numbers A barium enema in cases of amebic colitis may reveal an infiltrating lesion in the colon most often in the cecum which is due to an amebic granuloma The systemic forms of amebiasis include abscess of the liver brain lung and spleen Pleuritis pericarditis and ulceration of the skin may occur

Diagnosis is most often based on the results of repeated stool examinations A practical method consists of examination of purged stools immediately after they are passed on three successive days Shookhoff (32) has stressed the importance of well trained personnel to perform these stool examinations The complement fixation test as performed at the National Institutes of Health, is as yet only an aid in the diagnosis of amebiasis it should be employed when results of stool and sigmoidoscopic examination are persistently negative (33)

TREATMENT

The number and diversity of the available drugs and the frequent need for multiple courses of treatment to attain cure are evidence of the need for more effective amebicidal drugs than are now at hand In severe or moderately severe amebic colitis treatment should start with a daily dose of 0.06 Gm (1 gr) of emetine hydrochloride orally or better subcutaneously for seven days A similar course may be repeated after 10 days Emetine is a toxic drug and one should be alert for untoward systemic symptoms appearing during its use Emetine is supplemented by the administration of Diodoquin an iodoquinoline containing 63.9 per cent iodine which has proved reasonably effective and causes little intestinal disturbance Three tablets are given three times daily for 20

days Most (34) estimated a cure rate of 80 to 95 per cent for this drug To overcome the shortcomings of any one therapeutic agent it might be advisable to use multiple courses of more than one drug for example carbarsone in 0.25 Gm (4 gr) doses twice daily for 10 days following the course of Diodoquin (34, 35)

Although the antibiotics have proved effective in the treatment of amebiasis their routine use in the average case is contraindicated by their frequent untoward effects on the gastrointestinal tract These include nausea, diarrhea and anal pruritus

In a series of cases of acute amebic dysentery in Korea Martin *et al* (36) investigated combinations of amebicides (emetine, carbarsone, Milibis) and also the antibiotics They concluded that the most impressive results as gauged by initial response and lowest relapse rates were obtained by Terramycin in combination with one of the amebicides and also by Aureomycin combined with chloroquine diphosphate In a somewhat similar study in Venezuela Sanchez Vegas (37) found that the best over all results followed the combined use of the nonabsorbable bismuth glycolyl arsanilate (Milibis) 500 mg with the highly absorbable chloroquine diphosphate (Arlen) 150 mg twice daily for 15 days

BACILLARY DYSENTERY

The fundamental lesion in this acute infectious disease consists of ulceration of the mucous membrane of the large intestine The dominant symptom is frequent and painful diarrhea with stools containing or consisting solely of blood and mucus

Bacillary dysentery in man is caused by the *Shigella* group of gram negative bacilli They produce a potent toxin giving rise to severe symptoms which may prove fatal *Shigella paradysenteriae* and *S. sonnei* are less virulent than *S. dysenteriae* Dysentery bacilli multiply in the mucous membrane of the large intestine and cause inflammation and varying degrees of ulceration In mild cases this amounts to little more than desquamation of epithelium on the projecting edges of the folds of the mucous membrane In more advanced cases the lesions extend deeply and produce a series of

superficial ulcers having their long axes across the bowel. In severe infections there may be wholesale desquamation of mucous membrane with formation of large areas of highly inflamed granulation tissue covered by greenish black slough. Where extensive destruction of the mucosa has taken place regeneration does not occur and scarring results. If the condition becomes chronic lesions persist in the form of small irregular ulcers surrounded by inflammatory congestion.

SYMPTOMS—The stools which at an early stage are liquid and contain flakes of blood stained mucus soon become more frequent and in the course of a few hours consist solely of small quantities of blood stained mucopus. In a severe case the bowels move at least once or twice every hour indeed the desire to go to stool may be almost constant. There are severe griping and tenesmus and not infrequently considerable hemorrhage (38).

Dissemination is through contamination of food by unhygienic food handlers and by flies and occasionally through ingestion of water from a contaminated source.

Diagnosis is confirmed by stool culture early in the disease or by agglutination tests with specific serums which become positive in a dilution of 1:100 after a few days.

TREATMENT—The Joint Dysentery Unit, 64th Field Hospital, Far East Command treated 1,600 cases of known or probable shigellosis in Korea (39). Terramycin was found to be clearly superior to other antibiotics for all *Shigella* species. 2 Gm. initially and 1 Gm. 12 and 24 hours thereafter was a sufficient and effective dosage. Sulfadiazine was then given in an initial dose of 2 Gm. followed by 1 Gm. four times daily for four days. Clinical improvement was prompt within 24 hours the patient was relatively free of symptoms. Patients were released seven days after treatment if they had three negative cultures.

ANTIBIOTIC COLITIS

The extensive use of the broad spectrum antibiotics often results in side reactions characterized by immediate or delayed symptoms of

Specific Infections of the Anorectum

TUBERCULOSIS

FISTULOUS DISEASE of the anorectum has long been associated with the stigma of tuberculosis. This concept is erroneous. Fistulas excised from nontuberculous persons were proved to be tuberculous in only 1.8 per cent, and in these the disease was discovered only after careful screening by serial sections and guinea pig inoculations (1).

The incidence of anorectal fistulous disease in general hospitals averages 0.6 per cent of all admissions. Figures reported from a number of hospitals for the treatment of tuberculosis show that 5 to 11 per cent of patients with pulmonary tuberculosis have chronic anorectal fistulas and abscesses (2). At Serenissima Hospital, a New York City tuberculosis sanatorium, 6 per cent of all patients admitted have chronic anorectal inflammatory disease consisting of abscesses, fistulas, and chronic anal ulcers.

These figures clearly show that inflammatory disease of the anorectum occurs 10 times more often in patients with pulmonary tuberculosis than in the nontuberculous population. Furthermore, it has been conclusively shown that in patients with pulmonary tuberculosis histologic examination disclosed the anorectal inflammatory disease to be tuberculous in about 80 per cent. The infection probably has its onset in an anal crypt as a simple pyogenic infection which then goes on to abscess formation and fistulization. Sweeney

(3) was able to culture viable tubercle bacilli from smears taken from the washed rectal mucosa in 30 per cent of patients with pulmonary tuberculosis who had positive sputum. The tubercle bacilli constantly present in the feces of tuberculous patients are a potent source for tuberculous contamination of all open anal lesions, once contaminated the lesion spreads superficially and circumfer



FIG 95 (*left*) —Extensive perianal tuberculosis of serpiginous type incisions for drainage proved futile

FIG 96 (*right*) —Perianal tuberculous fistula

entially along the perianal lymphatic network and along the groin toward the inguinal nodes

Tuberculous perianal disease is seen in a variety of clinical forms. At Sea View Hospital chronic serpiginous subcutaneous abscesses (Fig 95) are seen most frequently. Characteristically these are widespread subcutaneous abscesses with multiple fistulous openings which exude very little pus. The overlying skin is indurated, edematous and of a livid dusky red color. Spread is along the perianal lymphatic network and is limited to the superficial tissues. In all patients with serpiginous subcutaneous lesions in our series biopsy specimens contained tuberculous granulation tissue.

Proctology

Perianal fistulas in tuberculosis patients are the sequel of acute pyogenic abscesses. They probably originate as simple pyogenic fistulas which later are contaminated with tubercle bacilli. The tuberculous fistula is characterized by a widely patulous irregularly serrated orifice surrounded by edematous, indurated violaceous skin (Fig 96).

Chronic anal fissures in tuberculous patients with positive sputum become tuberculous by contamination. The tuberculous ulcer ordinarily has elevated indurated edges and a base consisting of dark piled up granulation tissue. Lupus or tuberculosis of the perianal skin is seen in advanced toxic stages of the disease. Because of extensive concomitant involvement of the sphincter musculature it is considered unsuitable for definitive operation. Solitary tuberculids are rare, in the perianal region they are generally regarded as metastatic. They should be widely excised.

At Sea View Hospital the diagnosis of tuberculous perianal infection was based on the examination of biopsy material obtained at operation which showed histopathologic evidence of tuberculous granulation tissue i.e. typical tubercle formation with central necrosis fibroblastic capsule monocyctic infiltration and giant cells. Sweany (3) pointed out that failure of histopathologic diagnosis in tuberculous perianal lesions may be due to the fact that typical changes are present only in live granulations. When taking specimens the surgeon should attempt to remove intact areas containing live granulations.

To obtain even partial cure it is extremely important that tuberculous perianal lesions be recognized by their gross characteristics at operation. When the chronic lesions are surgically exposed typical tuberculous granulation tissue is seen in the base of the lesions. The granulations are cyanotic and almost violaceous friable and characterized by an absence of free bleeding when wiped away.

Perianal tuberculosis is a surgically curable disease. Martin (4) obtained cure in 87 per cent of 75 patients. Chisholm (41) cured 98 per cent of 71 patients. At Sea View Hospital 72 per cent of 68 patients with microscopically proved perianal tuberculosis were

cured 12 died of their pulmonary lesions while the anal wounds were not yet healed 10 per cent were discharged from hospital as arrested with unhealed anal wounds and 6 per cent remained with



FIG 97 —End result of six stage operation over 18 month period for extensive tuberculous perianal abscess and multiple fistulas: complete healing. (From *Granet Ann Surg.* 112 440 1940)

a persistent sinus One third of our patients required more than one operation one patient had six operations over an 18 month period before wound healing was complete (Fig 97)

TREATMENT

Management is basically surgical Acute abscesses are incised with stem to stem fistulectomy when possible Serpiginous abscesses are excised en bloc along with an adequate border of grossly normal skin and areolar tissue A tuberculous fistulous tract is excised en bloc Fistulotomy ■ not an adequate operation for tuber

Proctology

culous fistulas In widespread tuberculous perianal disease staged operations are advisable to insure support of the perineum by blocks of fibrous scar tissue between scars Uninvolved skin or subcutaneous tissue between fistulous or abscessed areas should be saved as these islands of normal tissue tend to maintain elasticity and minimize distortion due to scar contracture

When the internal opening is found to be placed above the anorectal levator ring operation is done in stages A seton is inserted through the internal opening and tied to encircle the sphincter muscles Excision of the fistulous tract is delayed until the external wound is healed with a firm scar adherent to the sphincter musculature

The successful treatment of surgical tuberculosis with streptomycin combined with para aminosalicylic acid (PAS) and more recently by the oral use of the isonicotinic acid derivatives (Rimifon and Nydrazid) has been extensively documented and requires no elaboration (4 5) These preparations have greatly improved the results in the surgical treatment of perianal tuberculosis It is our policy at Sea View Hospital to administer Rimifon or Nydrazid orally 5 mg per kilogram of body weight divided into two doses daily as supportive treatment Wide surgical excision is fundamentally the logical and best treatment for anorectal tuberculosis

LYMPHOGRANULOMA VENEREUM

This systemic infectious disease is caused by a virus related to that of the psittacosis group The disease commonly affects the anorectum Primary penile or vulval lesions are associated with secondary inguinal adenitis (bubo) or vulval esthiomene Lesions of the eye brain skin and spleen have been described

The fascinating history of this disease reflects the progress of scientific medicine through the years Nicolas Fivre and Durand described lymphogranuloma venereum as a distinct entity in 1913 Earlier observers had accurately described the rectal lesions but had considered syphilis gonorrhea or tuberculosis to be the cause of the stricture Freis description of the diagnostic skin test in 1925

gave further impetus to the study of the clinical manifestations of the disease and stimulated the search for reliable diagnostic tests by specific complement fixation and by skin tests. Bacon (6) discusses in detail the history of the disease and the development of diagnostic tests.

PATHOGENESIS AND CLINICAL FEATURES

The rectal lesion in its early stage is seen as an intense proctitis characterized by an injected turgid granular friable mucosa. It may show superficial erosions and there is a sanguineous mucoid exudate. Tenesmus, proctalgia and bloody stools are common. The intensity of the proctitis should suggest the diagnosis; specific diagnostic tests should be performed to establish it. The infection rapidly spreads to the submucosa where it persists as a diffuse and severe inflammatory exudative process with marked lymphocytic and plasma cell infiltration. Over a period of months extending into years progressive and continuous organization of this inflammatory exudate takes place. As a result the lumen of the rectum is narrowed over a rather extensive area developing finally into the characteristic tubular strictured rectum. During this prolonged period ulcerative proctitis of the mucosa persists and perianal lesions such as ulcers, abscesses, fistulas and esthiomene may ensue.

Despite the fact that the diameter of the lumen often contracts to 1 cm. patients seldom complain of constipation nor is fecal impaction common. The marked inflammatory proctitis probably stimulates increased secretory and motor activity of the colon with the result that stools are soft and readily pass the stricture.

The disease is transmitted to the rectum by direct implantation of the virus. Exhaustive studies in males attempted to correlate inguinal adenopathy as the earlier lesion with subsequent retrograde dissemination of the virus by the lymphatics and later infection of the rectum. Although conceivably contamination from the genital tract can occur in females it is becoming increasingly evident that rectal lymphogranuloma is most often a primary disease. Passive rectal intercourse is not uncommon in the social and racial

groups in which most cases of this virus disease occur. Grace and Henry (7) were able to elicit a history of exposure through passive rectal intercourse in 24 (63 per cent) of 38 males and in 5 (33 per cent) of 15 females with anorectal lymphogranuloma. They cited Bensaude who claimed this route of direct infection in 80 per cent of 78 males with the disease. Grace (7a) now feels in the light of his more recent experience that the latter figures are more nearly correct.

Rectal infection in children has been reported several times. The source of contamination was an infected mother who used her own enema nozzle for her children.

DIAGNOSIS

The Frei test when positive, is usually good evidence of the presence of the disease. As with many other biologic tests this test is not absolutely specific. A false positive reaction may be obtained in patients infected with the virus of psittacosis, and occasionally in patients with atypical pneumonia, ulcerative colitis and colorectal cancer. The Frei antigen was prepared originally from diluted and attenuated pus obtained from buboes. A highly sensitive skin test antigen made from specific virus grown in chick embryos is commercially available as Lygranum. The test is performed by intradermal injection of 0.1 cc. of Lygranum. The site is observed in 48 to 72 hours for reaction. A positive reaction consists of a reddish papule 6 mm. or more in diameter. A control test with uninfected chick embryo solution is run simultaneously to rule out allergy to egg. In patients who are clinically inactive but who at one time were infected with lymphogranuloma venereum the Frei reaction remains persistently positive. The complement fixation test prepared from an infected chick embryo antigen is highly sensitive in the active phase of the disease but becomes negative some time after cure. Bedson *et al.* (8) prepared an acid extract of lymphogranuloma virus and believe that higher specificity is obtained with this antigen.

TREATMENT

The response to treatment of rectal lymphogranuloma venereum depends on the particular stage of the disease. Patients with advanced stricture cannot be cured by conservative measures. Those with inflammatory proctitis and even ulcerative proctitis respond well to treatment with the sulfonamides and better still to broad spectrum antibiotic therapy. Complete clinical remission resulted in all of some 30 patients with simple or ulcerative proctitis at New York Hospital who were treated with 2 Gm. of sulfadiazine daily for several months. Lesions gradually resolved and strictures did not develop during a protracted follow up period. Early stages of rectal lymphogranuloma respond more rapidly to treatment with Aureomycin (9) and chloramphenicol (10).

Long standing disease with tubular stricture and ulcerative proctitis likewise responds well to treatment with the sulfonamides and antibiotics. However healing is limited to the proctitis and to a limited extent to the submucosal exudative process. Treatment progressively decreases the bloody purulent exudate to a point where it often ceases entirely. It has been shown particularly by Rowe (10) that the caliber of the lumen through the strictured area becomes wider following treatment. This increase in the diameter may well result from subsidence of edema and mucosal and submucosal inflammatory swelling as well as from the increase in mobility of scar tissue which takes place in old scars. Most patients with rectal lymphogranuloma venereum who come to the City Hospital Clinic (New York) have had the disease for years and have markedly contracted strictures with bloody purulent rectal discharge. The antibiotics are not available for these indigent patients and they are treated with sulfadiazine 2 Gm. daily in divided doses for three weeks of each month. With this regimen blood and pus diminish, there is subjective improvement and the lumen becomes slightly more patulous. In my experience the soluble sulfonamides are valuable for maintained treatment in advanced lymphogranuloma of the rectum.

Surgical treatment by colostomy for severe stricture does result

in subsidence of the inflammatory process. In advanced and complicated cases abdominoperineal resection of the rectum gives surgical cure. In carefully selected patients in good physical condition and without evidence of perianal infection, Breidenbach and Slattery (11) resect the involved rectum and re-establish function by a pull-through operation utilizing the transverse or descending colon. Of 21 patients thus treated results were good in 17, fair in one and failures in two, one patient was lost to follow up.

Pund and Lacy (12) found that 36 per cent of their Negro patients with anorectal and genital cancer had a positive Frei reaction. They believe that lymphogranuloma venereum may predispose to carcinoma, and that persistent lesions of lymphogranuloma should be considered precancerous. This concept is interesting particularly in view of Binkley's finding of positive Frei reactions in a number of his cancer patients.

GONORRHEAL PROCTITIS

In most patients symptoms are so slight that medical care is not sought. The possibility of gonorrhea as the cause of rectal burning, slight mucoid discharge and pruritus is often ignored by physicians and a smear is not taken for a Gram differential stain. Most patients are treated empirically and symptomatically, after a time, the organism loses its virility and spontaneous remission takes place.

The mucosa of the sphincteric rectum and the anal crypts are most often infected; the anus rarely. In genitally infected women contact contamination takes place readily. This is true also in children with purulent vaginitis which reveals gram negative diplococci on smears but rarely yields true gonococci on culture (13). Concomitant purulent proctitis is present in many children with vaginitis. Epidemics of gonorrheal proctitis in institutionalized infants have been reported.

In males gonorrheal proctitis almost always results from direct inoculation through rectal coitus. Finger contamination by a genitally infected female during heterosexual coitus has been described. Pederasty is not uncommon in adolescent boys and outbreaks of

rectal gonorrhea as well as anal syphilis have been reported in boys schools and in penal institutions (14) I have observed gonorrheal proctitis in overt homosexual males as well as two cases of acute rectal gonorrhea acquired by 16 year old boys at boarding school

Harkness (15) reported on 168 males with proved gonorrheal proctitis acquired by admitted sodomy Acute symptoms consisting of tenesmus burning and painful defecation were infrequent Mild symptoms manifested by anal moisture pruritus and verrucae occurred more frequently The greatest number 64 cases were symptomless The diagnosis in this group was made during examinations conducted during a search for venereal contacts

In acute and subacute cases an injected edematous friable mucosa is seen on endoscopy Purulent mucoid secretion is usually present between the columns of Morgagni or may be expressed from an anal crypt Subacute cryptitis is frequently present but abscess formation is rare The diagnosis is easily made by demonstrating the intracellular gram negative diplococci on a slide specimen of mucus obtained through the anoscope

In this antibiotic age treatment of gonorrheal proctitis is no longer a difficult problem

ANAL SYPHILIS

The presence of perianal condylomas as a manifestation of secondary syphilis is well known and requires no discussion here

Primary syphilis of the anus has not been extensively reported in the literature Its clinical manifestations are so bizarre that in most instances early diagnosis is missed or is incorrect The true nature of the lesion is revealed at long last by the appearance of remote secondary syphilids As is the case in rectal gonorrhea symptoms may be mild so that patients do not seek medical care Furthermore as with primary chancres in other locations the lesion heals spontaneously and the disease is missed in its primary phase Even if seen by a physician syphilis is not suspected and the lesion is treated as a benign anal fissure or ulcer

Martin and Kallet (16) in 1925 reported 20 cases of primary

anal syphilis found on rectal examination of 300 consecutive patients in a clinic attended largely by socially underprivileged individuals. A review of the literature has revealed only 16 additional reported cases of *anal chancre*. Of 11 patients with primary anal syphilis treated in the last five years (17), three were women and eight men. Seven were private patients. All but one patient admitted practicing passive rectal coitus. In one patient the specific nature of the lesion was not suspected until systemic manifestations of secondary syphilis appeared.

The most common primary lesion of anal syphilis is a superficial erosion frequently multiple with indurated edges and slight circumferential erythema. It is commonly misdiagnosed as an anal fissure or ulcer. The inguinal nodes on the side of the primary sore are enlarged and tender. Inguinal nodes are enlarged bilaterally in cases with contralateral kissing ulcers but may show no enlargement when the primary lesion occurs in the midline at the anterior or posterior commissure. The presence of inguinal adenopathy is an important diagnostic aid. Darkfield examination of the anal lesion often gives negative results due to secondary overgrowing contaminants. If inguinal nodes are enlarged the organism may sometimes be demonstrated by darkfield examination of material aspirated through direct needle puncture of the nodes. Results of serologic tests are rarely positive before the third week and are of no aid in the diagnosis of a primary lesion.

Primary chancre of the sphincteric rectum has been described as localized ulcers surrounded by indurated lobulated mucosa. Diagnosis was suggested by a history of rectal coitus and a positive serologic reaction. The lesion resolved completely following antisyphilitic treatment (18). Gumma of the rectum is rare. The lesion has been described as a smooth spheroid tumor with intact mucous membrane. A case described by Kallet (19) resembled an ulcerating carcinoma.

Lesions of anal and rectal syphilis respond promptly to adequate antisyphilitic treatment. All ulcerated lesions of the anorectum if bizarre in appearance if located laterally and if associated with

enlargement of the inguinal nodes, should stimulate a diagnostic search for specific etiology. One should think first of syphilis then chancroid and, finally granuloma inguinale. Furthermore it must be evident that in large cosmopolitan cities promiscuous homosexuality in males is widespread and is responsible for anorectal venereal disease.

ANAL CHANCROID

The specific lesion is caused by direct infection of the anal region with *Hemophilus ducreyi*; usually by sodomy. Incubation time is short—one to five days following exposure. The lesion, which may be single or multiple, is small with a superficial, rounded circumscribed ulcer which is painful. Inguinal nodes may be enlarged.

Direct smear may show chains of the gram negative bacilli. Ducrey vaccine for intradermal skin tests is commercially available. The result is positive if an indurated papule appears in 48 to 72 hours.

The lesions respond to local and oral therapy with the soluble sulfonamides. Streptomycin is reputedly effective in divided doses of 2 Gm. daily for three days.

GRANULOMA INGUINALE

The chronic ulcer of granuloma inguinale usually affects the genitals, the inguinal region and the perineum. Extension involving the anus may occur.

The lesion starts as a small papule which enlarges, ulcerates and spreads by contiguity over a period of months or years. The ulceration is covered by pink granulation tissue which is friable and bleeds easily (Fig. 98). As a part of the diagnostic procedure in chronic anal ulceration smears taken from the ulceration near its edge are searched for the pathognomonic Donovan bodies. With Wright's stain these appear as encapsulated stubby dumbbell shaped bacillary bodies grouped together in the cytoplasm of a large mononuclear cell.

Berkowitz (20) reported three cases of chronic ulceration in

volving the anus which proved to be granuloma inguinale. Two patients had constantly painful ulcerated indurated lesions at the anal verge which extended into the perianal tissues, in the third patient there was a nodular ulcerated mass at the anal verge which



FIG 98 (left) —Anal granuloma inguinale (From J Berkowitz New England J Med 234 625 1951)

FIG 99 (right) —Anal warts (verrucae acuminatae)

had its onset 10 years previously. In all three patients rapid healing followed specific treatment which consisted of intramuscular injections of Fuadin, an organic compound of antimony. Terramycin has been successfully used in the treatment of granuloma inguinale by Greenblatt (20a).

ANAL WARTS (VERRUCAE ACUMINATAE)

Soft filigreed condylomatous warts involving the anus and perianal region are frequently seen. Considered by all as a simple clinical

cal entity, their persistent recurrence after purported removal is disconcerting

PATHOGENESIS AND CLINICAL FEATURES

Many fanciful theories have attempted to explain the etiology of warts, but their exact origin is still unsettled Templeton (21) reproduced warts by intradermal injections of sterile filtrate material obtained from warts this filtrate presumably contained a virus Incubation time following inoculation may be four weeks to as long as 20 months The infectious nature of warts is indicated by their local dissemination through autoinoculation Their reappearance after removal is probably explained by their prolonged period of incubation

Anal warts (Fig 99) vary from 1 mm to 2 cm in size and from very few to so large a number as to surround the anus completely They extend into the anal canal but ordinarily their proximal spread is limited by the anorectal line They take origin from the papillary layer of the epidermis are usually pale salmon color and are friable The larger warts tend to become pedunculated There are no symptoms in the early stages but as the warts grow in number and size pruritus burning and pain appear An irritating discharge with a disagreeable odor is common Anal warts are most frequent in young men The association of lack of personal cleanliness with anal warts is described in the literature but I have not found this to be the case in most of my patients

TREATMENT

Successful eradication of anal warts depends entirely on frequent follow up examinations for at least six months after their initial removal It is not unusual for new warts to form a fortnight after all visible warts had been completely eradicated The newly formed warts must be removed immediately and repeatedly to prevent further autoinoculation

The technic of treatment is largely governed by the extent of involvement If there are only a few warts a most satisfactory office

treatment consists of removal of each wart by sharp excision with knife or scissors followed by electrodesiccation of the base. Anesthesia is obtained by skin infiltration with procaine followed by an anesthetic solution of prolonged action. An antiseptic ointment dressing (Triple Sulfa Cream) is maintained constantly at the anal region for several months during which the patient is examined every week at first and then every two weeks. For extensive lesions a similar procedure is carried out in hospital with regional anesthesia.

Local application of podophyllin has been advocated for the treatment of verrucae acuminatae (22). While it does indeed satisfactorily destroy the wart, my experience with podophyllin has been disastrous. The irritating resin could not be kept localized to the warty tumors for any length of time without its spreading to the perianal skin. The severe dermatitis caused by the podophyllin resulted in severe pain and swelling which necessitated treatment by bed rest, constant wet dressing and opiates. Because of these side effects podophyllin should not be used for the treatment of anal warts.

Dichloroacetic acid has essentially the same destructive action on anal warts as podophyllin without the disadvantages of the latter. This chemical cauterizing agent can be used as primary treatment instead of excision. The skin around the individual wart is protected by petrolatum and a drop of acid on a sharpened wooden applicator stick is carefully applied to the body of the wart. The acid is rapidly absorbed by the wart tissue and another drop is then applied. When this is absorbed the wart is sponged with water and smeared over with petrolatum. Other warts are similarly treated either at one session or at several sessions at short intervals. A burning pain follows application of the acid but subsides after about 10 minutes. Warts become desiccated and slough off in eight to 12 days. This treatment is very effective for individual warts which have reformed after excision.

No matter what treatment is undertaken the patient must be informed that re appearance of warts is to be expected over a long period of time. Cure can only be attained by frequent follow up

visits at which time the expected newly formed warts can be promptly destroyed

HIDRADENITIS SUPPURATIVA

This clinical and pathologic entity has its origin in one or more apocrine glands which become infected. The infection spreads by contiguity to involve adjacent glands, subcutaneous tissue and skin.



FIG 100—Hidradenitis suppurativa.

resulting finally in a chronic disabling widespread acneform and fistulous disease process (Fig 100)

The apocrine glands present in the hairy regions of the axilla, pubis, groin, perineum and perianal region do not function before puberty. They differ from the ordinary sweat glands (eccrine glands) in size, location and structure. The apocrine glands lie in the deep subcutaneous tissue well below the skin. Their over-all length approximates 7 mm, of which 4 mm constitutes the secretory portion and 3 mm the duct. The secretory tubules branch many times and are lined with cuboidal cells; the acini are filled with a granular material, the product of cellular disintegration. The orifice of the

excretory duct may be connected with a hair follicle or may empty into invaginated epidermis. Slowly growing neoplasms, termed hidradenomas have their origin in apocrine glands and may involve the perianal region (23).

The first manifestation of the disease is a localized nodule of induration in the submucosa. This may go on to local abscess formation characterized by induration and slow growth. Only a few drops of viscid pus are obtained on incision. Repeated episodes of single or multiple, acute or subacute abscesses follow, involving an ever widening area of contiguous apocrine glands. Subcutaneous sinuses form long tunnels joining adjacent infected areas. Fistulous orifices appear at the sites of the chronic apocrine abscesses. The skin of the entire area becomes thickened and brawny. The patient is subjected to repeated incisions of the abscesses and fistulas, all to no avail, for he continues to have a miserable existence with constant pain and discomfort from widespread purulent drainage.

Adequate treatment demands complete en bloc excision of the entire lesion in single or multiple stages (24-26). The involved skin sinuses and chronic abscesses are excised to the deep fascia of the perineum. When extensive denudation is necessary, contracture by excess cicatrization must be avoided by the use of skin grafts. In the perianal region, extensive resection of the entire lesion has proved a reliable curative procedure.

ACTINOMYCOSIS

Few cases of anorectal actinomycosis have been reported. Cope (27) collected 40 cases of which about half were primary in the rectum. Actinomycosis of the anorectum is rare in this country, most recorded cases were contained in foreign reports.

Actinomycosis is usually found involving chronic anorectal abscesses which are metastatic from actinomycosis elsewhere in the alimentary tract. Although the sulfur granules characteristic of actinomycosis may be found in the fistulous discharge, cultures from the growth reveal the causative organism to be the aerobic *Nocardia asteroides*. In true actinomycosis *Actinomyces bovis* can be cultured

from the sulfur granules. As this organism is frequently found in and around carious teeth, it is apt to pass through the gastrointestinal tract and contaminate mucosal defects at the anorectum. Gordon and DuBose (28) suggested this mechanism to explain the primary anal abscess in their case of actinomycosis of the anorectum, perineum and thighs of 30 years duration. The disease was apparently cured by intensive treatment with penicillin supplemented by sulfadiazine.

REFERENCES

- 1 Jackman R. J. and Buie L. A. Tuberculosis and anal fistula. JAMA 130 630 1946
- 2 Granet E. Treatment of perianal tuberculosis. Ann. Surg. 112 440 1940
- 3 Sweany H. C. Quoted by Martin C. L. Tuberculous fistula in ano. JAMA 101 201 1933
- 4 Martin C. L. In discussion on Skir I. *et al*. Streptomycin in the treatment of anorectal tuberculosis. Am J Surg 82 564 1951
- 4a. Chisholm A. J. Relationship of pulmonary tuberculosis to anorectal fistulae. Surg. Gynec. & Obst. 56 610 1933
- 5 Robitzek E. H. Selikoff I. J. and Ornstein G. G. Chemotherapy of human tuberculosis with hydrazine derivatives of isonicotinic acid. Quart. Bull. Sea View Hosp. 13 27 1952
- 6 Bacon H. E. *Anus Rectum Sigmoid Colon* (3d ed. Philadelphia J. B. Lippincott Company 1949)
- 7 Grace A. W. and Henry G. W. Mode of Acquisition of lymphogranuloma venereum of the anorectal type. New York State J. Med. 40 285 1940
- 7a. Grace A. W. Personal communication
- 8 Bedson S. P. *et al*. The laboratory diagnosis of lymphogranuloma venereum. J. Clin. Path. 2 241 1949
- 9 Fletcher A. *et al*. Aureomycin therapy in lymphogranuloma venereum. AMA Arch. Surg. 62 239 1951
- 10 Rowe R. J. Chloromycetin as an adjunct to management of lymphogranuloma venereum. Am J Surg 81 42 1951
- 11 Breidenbach L. and Slatery L. R. An operative procedure for lymphogranuloma venereum of the rectum. S. Clin. North America p. 399 April 1949
- 12 Pund H. R. and Lacy G. R. Jr. Lymphogranuloma venereum precipitating cause for carcinoma. Am Surg. 17 711 1951
- 13 Gradwohl R. B. H. *Clinical Laboratory Methods and Diagnoses* (St. Louis C. V. Mosby Company 1935)
- 14 Martin C. L. Rectal gonorrhea. Illinois M. J. 77 359 1940
- 15 Harkness A. H. Proc. Roy. Soc. Med. 41 476 1948
- 16 Martin E. G. and Kallet H. I. Primary syphilis of the anorectal region. JAMA 84 1556 1925
- 17 Granet E. and Kagan M. B. Unpublished data

- 18 Lieberman W Syphilis of the rectum Rev Gastroenterol 18 67 1951
- 19 Kallet, H I Proctologic aspect of syphilis Urol. & Cutan Rev 49 20 1945
- 20 Berkowitz J Granuloma inguinale A proctologic consideration New Eng Land J Med 234 625 1946
- 20a Greenblatt R. B, *et al* Terramycin in the treatment of granuloma inguinale J Ven Dis Inform 32 113 1951
- 21 Templeton H. J Long incubation period of warts Arch Dermat. & Syph. 32 102 1935
- 22 Lane C. W Verrucae a dermatologic problem J.A.M.A 144 1361 1950.
- 23 Cooper W. L. Role of apocrine sweat glands in the perianal tissue South M J 41 750 1948
- 24 Jackman R. H and McQuarrie J. L. Hidradenitis suppurativa Am. J Surg 77 349 1949
- 25 Christensen J. B. Hidradenitis suppurativa involving the para anal region Am J Surg. 79 61 1950
- 26 Conway H. *et al* Surgical treatment of chronic hidradenitis suppurativa Surg Gynec. & Obst. 95 455 1952
- 27 Cope Z. Actinomycosis involving the colon and the rectum J Internat. Coll. Surgeons 12 401 1949
- 28 Gordon M. A and DuBoise H. M. Anorectal actinomycosis Am. J Clin. Path 21 460 1951

Pruritus Ani

INTENSE ITCHING at the anus *pruritus ani* is a condition having specific characteristic symptoms despite multiple causes. It is chiefly this confusion between the symptom and its numerous origins that has led to great diversity in its management. Scarborough (1) reviewed the literature on *pruritus ani* and found etiologic theories that included such widely divergent concepts as inheritance of neurologic characteristics, outlets for emotional stress, pyorrhea and foci of infection in the teeth and tonsils. Many constitutional conditions including hepatitis, pancreatitis, diabetes, tuberculosis, and senility are occasionally associated with generalized pruritus. Anal itching when it occurs in these conditions is rarely an outstanding symptom.

In practice, most patients who present themselves with *pruritus ani* as the chief complaint have had the condition for a protracted time, have run the gamut of numerous home remedies, salves, and suppositories, and have been treated by various medical, physical, and surgical measures. In a personally studied series of 100 patients, 17 had had severe *pruritus ani* for 10 to 40 years. In a review of the history of these sufferers, it was noteworthy that after an interval of freedom from itching following some presumably specific form of treatment (x-ray, nerve block, or surgery), symptoms reappeared. Recurrences and remissions occurred in all patients studied in this series, irrespective of the specific therapy used. The proponents of nerve block by injection of alcohol or of the oil-soluble or deposi-

tory anesthetics make no claim for permanence of relief. Although remission of symptoms has occurred after roentgen therapy, recurrence is a constant feature in severe cases. Nerve section by various undercutting operations also afforded only temporary respite. A number of patients had hemorrhoidectomies, excision of tags, cryptectomies, and papillectomies; despite these plastic procedures recurrences brought the patient again to seek relief for intractable itching.

Thus, despite the various treatments for pruritus ani, none gave permanent cure. So far as can be determined from the literature, no author is confident enough to report successfully maintained cures in all patients treated by his method. An obvious constantly present factor which is mentioned only casually by many authors is anal hygiene and cleanliness. Contamination of the anus and perianal skin by feces after defecation is a constant feature in all patients, and is also the one factor which persists following medical, physical, or surgical treatment of pruritus ani.

ETIOLOGY AND PATHOLOGY

Rothman (1a), who investigated the physiology of itching, described a state of increased itching excitability persisting in an area surrounding the primary site of the itch response to a skin irritant. In these itch sensitized areas the threshold for itch stimuli is lowered so that light touch, heat, or scratching, which do not ordinarily invoke itching, incite violent pruritus. Scratching relieves itching. In the itch sensitized anus, however, even the act of desisting from scratching can be the trigger mechanism releasing the itch impulse. As a result of this mechanism, the itch-scratch-itch cycle continues indefinitely, resulting finally in epidermal lichenification, fissuration, and cellulitis (7). The anus and perianal region are richly endowed with itching points, as shown by Longo (Fig 101).

Tucker and Hellwig (2), in a significant study, emphasized the importance of fecal contamination in pruritus ani. They studied specimens of skin from the anal verge removed at biopsy from 13

patients in various stages of the disease. They commented on the similarity of the histologic picture seen in the early stages of chronic pruritus ani (Fig 102) and in the pathologic lesions of the skin in cases of chemical dermatitis. They stated: Hydrops of the epidermis cells, irregular proliferation of the stratum mucosum and of the hair follicles, hyperkeratosis with plugging of the hair follicles and atrophy of the sebaceous glands (seen in pruritus ani) are changes



FIG 101—Relative density of itching points around anorectum (From A. L. Shapiro and S. Rothman, *Gastroenterology* 5:155, 1945.)

characteristic of dermatitis due to chemical irritants. Furthermore, biopsy specimens from within the anal canals showed no pathologic lesions and resembled the histologic picture of specimens from the anal canal of 345 patients without pruritus ani. They argued from this that although anal and rectal disease may exist in patients with pruritus ani, the anal lesion itself is not the essential cause of the pruritus. They concluded that pruritus ani seems to be a simple chemical dermatitis due to something in the feces and suggested that the irritant may be skatole or some similar decomposition product of proteins. These observations coincide with the clinical experience of other investigators. With careful anal hygiene, most patients with mild pruritus ani can remain symptom free.

The secretions found on the rectal mucosa of most individuals

are highly alkaline. In approximately 1 000 patients tested with nitrazine paper applied to the mucosa during routine endoscopic examinations the reaction was uniformly alkaline. Values below



FIG 102 —Perianal skin in chronic pruritus ani early stage note hydrops of cells of malpighian layer (From C. C. Tucker and C. A. Hellwig Arch. Surg. 34 929 1937)

pH 6 were obtained in only 38 cases. In patients with chronic anal pruritus the rectal contents were found to be of abnormally high pH (8 to 10). Bacon and Hardwick (5) have called attention to the chemically irritating action of strong alkaline rectal contents on the perianal skin as an etiologic factor in pruritus ani.

It is evident that many physicians do not seriously attempt to

determine the etiologic factors in their patients with pruritus ani but direct their therapy empirically to the symptom. In the absence of a common etiology there can be no uniformly adequate treatment.

One constant important factor is present in all cases of pruritus ani: the continued fecal soiling of the perianal skin. In certain atopic individuals sensitivity to chemical substances in their own feces by constant contact probably results in perianal dermatitis with itching as the chief symptom. Furthermore the perianal skin is naturally well adapted to the development of contact dermatitis since heat, moisture, skin folds, apocrine secretions, and hair are all present. Added to the constant mechanical friction of walking or sitting is the act of using toilet tissue after defecation. This is tantamount to inunction of feces into the perianal skin. Feces contain numerous chemical substances that may irritate the skin. Among these are bacteria and their exotoxins, intestinal enzymes, and also the end results of bacterial action on ingested proteins—the indoles, phenols, and skatole. In a number of patients with severe pruritus ani, solutions of indole, skatole, and fecal emulsions applied in patch tests in the axillary region (which closely approximates the perianal region in its physical and chemical characteristics) produced a dermatitis.

Inflammatory lesions in the anal canal and lower rectum: proctitis, hypertrophied papillae, infected anal crypts, mucosal fissuration, chronic fistula, or anal fissure may cause anal pruritus. All of these lesions give rise to maceration of the perianal skin by constant soiling with irritating pus and by actual inflammatory changes in the sensory end nerves in hypertrophied papillae and excoriated perianal skin.

Perianal skin lesions frequently associated with pruritus are due primarily to mechanical abrasions caused by irresistible scratching. In many patients the scratching is done during sleep. Occasionally cellulitis complicates the primary pruritus and aggravates the problem of treatment.

The concept that patients with obstinate pruritus ani are specifically sensitive to a chemical substance or substances in their own

feces forms the basis of treatment in most cases. I believe that this relationship exists and is an important causative factor in all cases except those due to food allergy, and to mycotic or other specific dermatologic entities. Obviously pathologic lesions such as fissures, fistulas and cryptitis when present, are excepted as is pinworm infestation. Excepted also is the occasional patient with a psychoneurotic personality.

In the management of patients with pruritus and a detailed history of previous treatment, remissions, and especially the relationship of itching to defecation and its occurrence after retiring is of value. Constitutional disease must be ruled out by physical examination and laboratory investigations. A complete rectal examination is mandatory. Mucosal prolapse is commonly found on anoscopic examination, it is an important causative factor in fecal soiling of the perianal skin because with sitting coughing straining or passing flatus the prolapsing mucosa can extrude rectal mucus or feces through the anal sphincter. In many patients with pruritus residual feces are found in the lower rectum despite the fact that defecation had occurred shortly before anoscopic examination. These patients have learned by personal experience that an episode of severe itching can be temporarily abated by a cleansing rectal lavage.

DIFFERENTIAL DIAGNOSIS

In some patients, the lesions of the perianal skin are definite dermatologic entities. The most frequent of these conditions are the mycotic lesions evidenced by a circle of sodden thickened epidermis extending outward from the anus and often upward between the gluteal folds. Because of secondary bacterial contamination it is difficult to culture the fungi. In a carefully studied series of patients with pruritus and extending over 13 years Castellani (3) was able to demonstrate epidermophytosis in 20 per cent. Terrell and Shaw (4) reported similar results. Patients with perianal skin lesions due to fungus infections frequently have associated mycotic interdigital lesions, tinea cruris of the groin or phytid eczematoid lesions on the hands. Lichen planus can occur in the perianal region. It is distinguished by its violaceous patches of small polyangular flat topped

lesions with little or no pruritus. These lesions yield promptly to roentgen therapy.

Psoriasis which occasionally occurs in the perianal region presents a sodden beefy appearance. In these cases, diagnosis is clarified by typical psoriatic lesions elsewhere. Circumscribed neurodermatitis may appear in the anal region and is characterized by a dry, lichenified, infiltrated lesion. It is highly resistant to treatment as is well known. Enterobiasis (pinworm infestation) a common cause of intense anal itching is most frequent in children although not at all rare in adults. It should be sought for routinely in all cases of pruritus ani. Depending on the etiology in the individual case proper therapy conscientiously administered and utilized indefinitely by the patient will afford relief if not cure in most instances.

TREATMENT

After reviewing the vast literature on the treatment of pruritus ani one is impressed with the unreasonableness the utter lack of specificity of the measures advocated and in many instances the intricate technic for their fulfilment (7-9). For example tattooing of the perianal skin with mercuric sulfide has been reported as efficacious in uncomplicated pruritus ani. Nevertheless the method has not been generally adopted possibly because of the difficulty of its application (10).

It would be futile to attempt a critical evaluation of all the methods of therapy advocated for pruritus ani. The history obtained from any patient with pruritus ani of long standing is in itself a condemnation of the therapeutic armamentarium in this disease. The principles of therapy which have long afforded satisfactory relief for many of my patients with pruritus ani will be described as well as the rationale for their utilization. Emphasis in therapy should be placed on simple measures which patients can understand and will persist in applying so that relief from their intolerable itching can be maintained even though permanent cure may not be possible.

ANAL HYGIENE—Treatment is directed mainly to keeping the perianal skin free of feces. Significant pathologic lesions of the ano

rectum must be eliminated by surgery. Secondary skin infections, such as abrasions from scratching cellulitis and skin and mucosal fissuration must be treated by measures calculated to restore a normal healthy skin. The patient must be made to understand that there is a permanent and direct relationship between perianal soiling with feces and the itch. Furthermore, it must be emphasized that successful treatment depends on the patient's careful cooperation in following instructions implicitly, and that the full responsibility for success or failure of treatment is his. Instructions in the care of the perianal region are carefully explained and the following printed directions are given him:

After defecation whenever possible take a rectal enema using warm tap water. Expel this immediately. A convenient method is with a 2 oz. rubber hand syringe (infant's enema syringe). Cleanse the perianal skin with wet absorbent cotton. Do not use toilet tissue. Dry well with cotton and powder with talcum. Cleansing with wet cotton and dusting with powder must be repeated about four times daily depending on the amount of moisture and the degree of itching. Keep the skin around the anus always clean and dry. Carry cotton and talcum in separate envelopes in a pocket or handbag. Before retiring repeat the small enema and cleanse dry and powder the anal region. After retiring rub a small amount of ointment well into the perianal skin. This may cause burning for several minutes.

Rectal lavage is intended to remove residual feces from the lower rectum and anus following defecation and is recommended before retiring because in most patients the itch is intensified at night. During the day feces may accumulate in the lower rectum and its removal before retiring frequently prevents nocturnal itching. The use of mineral oil is interdicted. I have found that oil mixed with feces frequently leaks through the anus to soil the perianal skin.

The ointment prescribed is half strength Whitfield's ointment modified as follows:

Menthol	0.2
Phenol	0.5
Salicylic acid	1.0
Benzoic acid	2.0
Acid Mantle Creme (Dome)	60.0

This preparation is used routinely for three reasons (1) Mycotic infections are associated with pruritus ani in about 20 per cent of all patients (3) and on an empiric basis routine use of antimycotic medication is advisable (2) The ointment usually causes moderately severe burning for some 10 minutes after it is applied to the perianal skin. The discomfort is accepted gladly in preference to the intolerable itching that plagues the patient on retiring. Furthermore the burning removes the necessity for scratching. Abrasions and reinfection of the perianal skin are thus prevented the vicious cycle is broken and the excoriated infected skin tends to heal rapidly (3) Acid Mantle Creme contains aluminum acetate in a hydrophilic (vanishing cream) base which has a pH of 4.2. Its astringent protective effect on the skin is augmented by its acid reaction which somewhat neutralizes the excoriating alkaline mucus secreted by the rectal glands of Lieberkuhn.

Patients with severely inflamed and excoriated skin are treated with applications of wet dressings (*see* Chapter 4). These dressings are applied overnight under a perineal pad. In very severe cases of dermatitis with cellulitis bed rest for several days and constant applications of wet dressings are necessary. Treatment with ointment is withheld until the condition of the skin has improved but rectal lavage and perianal cleansing are used as described.

It has been my experience that with increasing clinical improvement patients voluntarily desist from rigid treatment so that cleansing throughout the day is omitted and rectal lavage is discontinued. Pruritus recurs in most patients soon thereafter. A survey was made of 80 patients with chronic and severe pruritus in order to evaluate the effect of therapy consisting solely of this simple form of anal hygiene. After several weeks of this management 93 per cent of patients reported satisfactory alleviation of the pruritus. The best results were obtained in the more cooperative patients regardless of the amount of skin change or the duration of the disease. Of the 50 patients who returned for follow up examination six months after discharge from active treatment 40 reported recurrence of the pruritus when the treatment was stopped or after an interval of careless

management Only 10 patients remained completely well This study tends to confirm the direct relationship of fecal perianal soiling to pruritus ani (11) Particularly striking was the prompt recurrence of symptoms when anal hygiene was discarded

ADJUNCTS TO TREATMENT—In addition to the basic treatment with anal hygiene, both systemic and local measures are utilized Most patients with pruritus ani are high strung sensitive vagotonic individuals who cannot tolerate even mild physical discomfort Many have several soft excoriating stools daily Nervous tension is increased in both men and women during the climacteric As individuals differ in personality and habits therapeutic measures must be suited to the make up of the individual as well as to the local pruritic lesion The diet is not generally disturbed except in patients with suspected allergies Chocolate eggs shellfish and other frequently allergenic foods are best discontinued Strong condiments are banned, spirits are allowed only in minimal quantities well diluted Cocoa or tea is substituted for coffee if the patient has frequent stools If loose stools persist 5 minims of deodorized tincture of opium on arising is prescribed in an attempt to limit defecation to one stool daily Yogurt $1\frac{1}{2}$ pt daily and buttermilk several times daily are advised, these foster the growth of *Lactobacillus acidophilus* (12) On this regimen maintained for several weeks rectal pH of 8-9 has been reduced to 6-7 as determined by repeated tests with nitrazine paper

Because many patients have severe pruritus after retiring or are awakened from sleep by the itching 100 mg of Benadryl or some other antihistamine is advisable at least an hour before retiring The soporific side effect frequently associated with Benadryl is valuable in inducing and maintaining sound sleep If itching is severe during the day an antihistamine with minimal side effects should be used

Women in the climacteric with symptoms of vasomotor instability should be given estrogenic therapy in proper dosage Testosterone is helpful for elderly men with vasomotor and emotional disturbances

In high strung individuals it may sometimes be necessary to

start with measures which will first and quickly, allay the intolerable itch. Roentgen therapy almost always gives quick relief, but extreme caution is in order because of the deleterious effects of x rays on fertility. Subcutaneous infiltration of the perianal skin with a long acting anesthetic agent will relieve some patients temporarily. Cellulitis is a contraindication to the use of these agents. The anal hygiene routine is instituted while the pruritus is in abeyance and skin fissuration and excoriations are healing. If these indi-



FIG 103—Subcutaneous neurotomy dotted line indicates area undercut.

viduals continue the regimen of anal hygiene recurrence of severe pruritus is unlikely.

Anorectal traumatizing lesions such as chronic fissure, prolapsing hemorrhoids, severe cryptitis, and rugated thickened skin tags must be excised. In addition, it is almost always advisable to extend the scope of the operation to include a perianal subcutaneous neurotomy. This procedure, which assures relief from pruritus during the postoperative period, consists of dividing the sensory nerves to the epidermis of the anus and the perianal skin in the pruritic zone (Fig 103). This is performed simply by incising the perianal

skin 2 cm beyond the pruritic zone at the NE NW SE and SW points of an imaginary compass. Through each incision the skin is separated from the subcutaneous areolar tissue by dissection with curved blunt-end scissors. The perianal skin is undermined completely around the anus and the anal epithelium is freed up to the anorectal line. Rather free bleeding from severed venules and arterioles results, but is readily controlled by a pressure dressing. The skin is kept separated from the underlying areolar tissue by loose packing with hemostatic Gelfoam or Oxyel gauze placed under the skin flaps. Anesthesia of the perianal skin and freedom from pruritus persists for four to six weeks postoperatively. The routine of anal hygiene is instituted during convalescence and is maintained indefinitely after complete healing of wounds.

My experience with varied forms of treatment has been that recurrences are frequent and that severe complications may follow certain therapeutic measures. Two patients had perianal abscesses after subcutaneous injection of anesthetic oil solutions and an extensive abscess followed alcohol injection by the Stone technic. Huhner (6) reported six cases of azoospermia following roentgen treatment administered by competent dermatologists for dermatoses of the perianal region; four of the patients were known to have had active spermatozoa before treatment. The testicles had been protected in all but apparently insufficiently. I have seen two cases of azoospermia after x ray treatment for pruritus ani.

NEUROGENIC PRURITUS ANI

In the average case of pruritus ani simple therapy conscientiously and continuously applied by the patient will afford relief if not cure in most patients. But in an occasional individual with persistent anal pruritus no adequate cause can be demonstrated despite the most painstaking investigation. These patients continue to have symptoms even when the gamut of our therapeutic armamentarium has been exhausted. It is noteworthy that the perianal skin of many such patients shows little evidence of the usual chronic dermatitis or lichenification though it may show self induced scratch

marks. Except for the latter the perianal skin appears essentially normal in all respects. Three such patients were women with personality difficulties, all had anal pruritus of long standing with little or no perianal dermatitis. The pruritus remained persistently recalcitrant to a routine of therapy which proved highly successful in allaying symptoms in other patients. Turell (10) records therapeutic failures in similar cases.

Persistent anal pruritus without demonstrable etiology in a patient with relatively normal perianal skin and who has personality difficulties or neurotic conflicts must of necessity be classified as a neurogenic pruritus ani. Numerous reports of this psychosomatic disturbance have been published. It is becoming increasingly clear that in certain neurotic individuals the anal pruritus may be the manifest subjective symptom derived from subconscious tension states. The definite pleasure relationship of pruritus ani and the scratching is brought out by Drueck (13) who wrote that the occurrence of itching in an apparently healthy skin as an expression of fixation phenomenon arising from a submerged or repressed anxiety or desire is familiar to both dermatologists and neurologists. The substitution of the pleasure of scratching an itching dermatitis for the pleasure of sexual orgasm is described by Sack as '*organistic pruritique*': the patient creating the itch in his own mind so to speak, in order to have the pleasure of gratifying his sexual desire by scratching it perhaps without any dermatic foundation.

Rosenbaum (14) reported two cases of neurogenic pruritus perinei and concluded: 'It is well known that the sensation of itching with its resulting scratching may result in pleasant and sensuous sensations at times actually resulting in sexual orgasm while at other times (or at the same time) these symptoms may produce intense suffering with violent scratching so that the end result amounts to complete self-destruction tearing oneself to pieces.'

Saul (15) described the case of a young man who complained of occasional severe attacks of pruritus ani. In the psychoanalytic material it became evident that the pruritus occurred regularly on occasions when he would be taken out by older men who were

interested in him. The analysis showed clearly that passive anal homosexual wishes were aroused by these situations. He occasionally indulged in anal masturbation and stated that he often used the pruritus merely as an excuse for this indulgence.

It is important that we recognize patients with neurogenic pruritus ani. Local therapy in such cases is futile except as a means of substituting a treatment ritual for the impulse to scratch. The elaborate treatment ritual of anal hygiene already described is often efficacious, as it satisfies the need demanded by certain psychoneurotics for a compulsive mechanism. In severe cases however, psychotherapy should be utilized without delay. A case in point is that of a 28 year old man with a two year history of intractable pruritus ani, treated intensively and unsuccessfully with x rays, ultraviolet light, and sundry subcutaneous injections who was hospitalized because of severe perianal cellulitis induced by scratching. Following prolonged therapy with continuous wet dressings, bed rest, sedatives, and antibiotics, a modified Ball undercutting operation (subcutaneous neurotomy) of the perianal skin and anal epithelium was performed. Within 10 days after operation severe anal pruritus recurred despite complete anesthesia of the undercut area to touch, pain and temperature—proof that his fixation phenomenon was in no way related to the physiology of nerve healing. Incidentally, this young man was found to have azoospermia. That this condition probably resulted from excessive x ray treatment is indicated by the fact that he had two children, the younger aged 17 months.

Many patients with so-called intractable pruritus ani are anal erotics (16). Their itch is the manifest subjective symptom derived from their hidden subconscious conflicts. Like all subjective symptoms of neurotic origin it has a distinct value in the emotional economy of the neurotic individual. He is loathe to give up the symptom of intense itching with its pleasure-pain component and retains it despite all local treatment. In practice we must recognize and suspect such cases. Much time would be saved by adequate psychotherapy early in the disease.

REFERENCES

- 1 Scarborough R A Pruritus ani Its etiology and treatment *Ann Surg* 98 1039 1933
- 1a Rothman S Physiology of itching *Physiol Rev* 21 357 1941
- 2 Tucker C C and Hellwig C A Pruritus ani Histologic picture in forty three cases *Arch Surg* 34 929 1937
- 3 Castellani A Pruritus ani and pruritus vulvae of mycotic origin *Practitioner* 117 341 1926
- 4 Terrell E H and Shaw F W Observations on fungal infections of the perianal skin and rectum *South M J* 21 887 1978
- 5 Bacon H E and Hardwick C E Pruritus ani A biochemophysiologic entity *J M Soc New Jersey* 44 446 1947
- 6 Huhner M Sterility and the x rays *JAMA* 104 1808 1935
- 7 Shapiro A L and Rothman, S Pruritus ani A clinical study *Gastroenterology* 5 155 1945
- 8 Bodkin L G Amino acid therapy for pruritus ani *Am J Surg* 82 557 1951
- 9 Matt J G Treatment of idiopathic pruritus ani with adenosine 5 monophosphate *South M J* 44 537 1951
- 10 Turell R Tattooing with mercury sulfide for intractable anal pruritus *Surgery* 23 63 1948
- 11 Granet E Pruritus ani The etiologic factors and treatment in 100 cases *New England J Med* 233 1015 1940
- 12 Seneca H Henderson E. and Collins A Bactericidal properties of yogurt *Am. Pract* 1 1253 1950
- 13 Drucek C J Essential pruritus perinei *J Nerv & Ment Dis* 97 528 1943
- 14 Rosenbaum M Psychosomatic factors in pruritus *Psychosom Med* 7 52 1945
- 15 Saul L J Incidental observations on pruritus ani *Psychoanal Quart.* 7 336 1938
- 16 Granet E and Hammerschlag E Anal eroticism and certain anorectal syndromes *Rev Gastroenterol.* 16 549 1949

Proctalgias and Anorectal Dyscrasias

PAIN OR DISCOMFORT in the perianal region and rectum is most often due to conditions associated with inflammation, infection or neoplasms. The anorectal symptoms to be considered here are of interest because each poses a problem in etiology, pathophysiology, and treatment.

COCYGYDYNIA

In this syndrome the pain usually begins as a sense of weight or heaviness which first involves the low back region. The discomfort gradually becomes more severe and progresses to an aching pain centering in the coccyx or sacrum frequently radiating up into the rectum laterally into the buttocks and occasionally down the thighs. The characteristic pain is severe continuous throbbing and is commonly brought on by prolonged sitting. Trauma resulting from falls on the sacrococcygeal region or associated with parturition, anorectal inflammatory conditions or anorectal surgical procedures is responsible for the onset of this syndrome. Bony lesions of the coccyx itself following trauma are infrequent according to Duncan (1). He found that in patients with coccygodynia studied at the New York Orthopedic Hospital fracture of the coccyx occurred in only 4 per cent and dislocation in 2 per cent of 262 patients.

The usual symptomatic treatment with sedation, physiotherapy, injection of analgesic solutions around the coccyx and even coccygectomy have too frequently given little or no relief. Based on his

Careful observations in 169 patients over a 16 year period Thiele (2) believes that the pain in this condition results from tonic spasm of the pelvic muscles which insert into the margins of the coccyx and sacrum. He reported that as early as 1859 Sir J. Y. Simpson described this mechanism by calling attention to the fact that when the coccyx or the coccygeal joints have been injured or when the surrounding tissues were the seat of inflammation any contraction of the muscles attached to the coccyx would excite the characteristic pain of coccygodynia. Thiele demonstrated the definite relationship between the coccygodynia and tonic spasm of the levator ani and coccygeal muscles and in some cases of the piriformis muscle. He found that levator spasm resulted from anal infection in 63 per cent and from trauma (falls, parturition, long train or automobile rides) in 29 per cent and followed anorectal surgery in 6 per cent of 169 patients with coccygodynia. In patients personally treated by Thiele the physical findings were described as follows. On digital rectal examination with the patient in the Sims's position spasm of the levator and coccygeus is easily detected by lateral posterior pressure the spastic muscles being felt stretched from their origin at the arcus tendineus or ischial spine to the side of the coccyx and the lower part of the sacrum. Piriformis spasm is difficult to detect because the distance of this muscle from the anus makes palpation uncertain.

My own experience (3) in a considerably smaller series completely confirms Thiele's findings. However another finding to my knowledge not previously described was noted in many of my patients the presence of well developed bursae which on finger palpation were felt in the lateral pelvis between the pubococcygeus and the ileococcygeus portions of the levator ani. They averaged about $0.5 \times 1 \times 2$ cm. were flat oval crepitant and exquisitely tender during the acute phase. It is readily conceivable that levator bursitis resulting from injury or infection can cause severe spasm of the adjacent muscles with constant forward traction on the coccyx resulting in rectal and low back pain. The prompt relief obtained

by specific local treatment is proof that the mechanism of pain in this syndrome is indeed chronic muscle spasm.

The definitive treatment of the coccygodynia syndrome is directed to alleviating muscle spasm. The technic originally described by Thiele places the patient on the table in Sims's position with the operator's gloved finger inserted full length into the rectum. Thiele stated:

Latero posterior pressure will place its flexor surface horizontally across the surfaces of the levator ani and coccygeus muscles almost at a right angle to their fibers. These muscles are massaged in the long direction of their fibers in the same manner that a strip is stroked by a razor. Massage is begun lightly. This is necessary because one does not wish to traumatize the extremely tender spastic muscles. As the patient makes subsequent visits massage is made with increasing pressure.

About two minutes of active massage to each side is adequate. Massage is followed by short wave diathermy applied to the sacral region with sedative heat for about 20 minutes. This technic is likewise utilized when acutely inflamed bursae are present.

Improvement is rapid with considerable relief frequently resulting from the first treatment. My patients are treated every other day for about six treatments; intervals are then lengthened until there is complete freedom from symptoms. If no improvement results after a few treatments the case is considered a therapeutic failure for this method. Of Thiele's 169 patients, 63 per cent were cured, 27 per cent improved and approximately 7 per cent were not benefited. When anorectal infection is present these foci should be removed surgically. A number of patients in whom conservative treatment failed had refused necessary surgical eradication of infected foci.

In my own experience with these methods, patients with tender inflamed bursae did well on massage; the size and tenderness of the bursae subsiding with clinical improvement. The few patients who do not respond to massage treatment and have no demonstrable anorectal foci of infection are given a trial of injection treatment with an oil soluble anesthetic agent. The solution is distributed into

the portion of the levator muscle adjoining the coccyx, 5 cc being injected on each side. With the index finger supporting the muscle from within the rectum the needle is passed through the subcutaneous tissues adjacent to the coccyx and fanned out into the muscle guided by the inserted index finger. After the injection the oil is distributed by massage of the muscle. The period of muscle relaxation following anesthesia persists for about 10 days, during which time massage and diathermy are instituted.

Coccygectomy is indicated when the coccyx is markedly deformed or everted following severe dislocations or fractures.

PROCTALGIA FUGAX

This term introduced by Thaysen (4) in 1935, is applied to a syndrome of idiopathic rectal pain with specific characteristics and of occasional occurrence. Perhaps credit for the original clinical description of the syndrome belongs to MacLennan (5) who in 1917 reported two typical cases of the syndrome under the title *Rectal Crises of Non Tabetic Origin*.

Although the clinical picture varies a composite description of the symptoms in a number of my patients would read somewhat as follows. Onset is insidious without warning often awakening the patient from a sound sleep in the early hours of the morning. There is a sense of discomfort in the rectum localized some 5 to 10 cm above the anus which increases rapidly in intensity to become a severe cramp-like pain often associated with mild shock. In its most severe form it is described as an agonizing localized pain as if caused by a constricting band. The paroxysm persists at maximum intensity for some five minutes then gradually subsides leaving the patient with a feeling of pronounced weakness and fatigue. With the resulting relief the patient speedily falls into a sleep of exhaustion. The anus itself is not primarily involved although secondary voluntary spastic muscular contractions are common. Patients attempt to relieve themselves of the pain by attempts to pass gas or defecate by hot baths, enemas and various changes of position with little effect on the condition.

Proctology

The actual cause of the pain is not known Karras and Angelo (7) discussed etiologic factors thoroughly in their complete survey of the syndrome

That the piroxysm of proctalgia fugax is caused by severe, tonic muscle spasm is indicated by such clinical features as specific localization to the same area in the rectum gradual increase in intensity with an agonizing maximum of a few minutes, and gradual subsidence probably as a result of physiologic muscle fatigue The pain of this syndrome is sometimes reported by patients as occurring directly following defecation In these instances, however the cramp is less severe in both intensity and duration From the location and intensity of the pain it is conceivable that the powerful puborectalis portion of the levator ani muscle which encircles the sphincteric rectum may be the culprit

The transient unpredictable character of the attacks affords little or no opportunity to study physical features or the physiology of the condition Most reports state that attacks occur once to three times a year The infrequency of the attacks and their almost invariable occurrence in the night make vigorous therapy impractical and difficult to evaluate while the unpredictable onset and infrequency of the attacks prohibit prophylactic measures The probable psychosomatic nature of this disorder is suggested by its onset in sleep often during periods of tension states (fear guilt fatigue) and its frequent onset following coitus masturbation and other phases of excited sexual states (3) The following history strongly suggests a psychosomatic genesis concentrated and conflicting feelings of rage pleasure guilt and retribution cause the strong retention phenomenon spasm and pain

A tense high strung 43 year old woman complained of nocturnal attacks of spasmodic rectal pain of many years duration. She was born with an imperforate anus which was corrected by surgery two days after birth. Throughout her childhood and adolescence the relative incontinence and fecal soiling were a source of great concern fear and embarrassment as well as punishment administered by unsympathetic parents The natural result was an exaggerated concern over her anus really an anal fixation A further significant factor is that operative necessity placed the anal opening at the

posterior verge of the labia a situation which favored genitalization of the anus. Periods of mucous diarrhea followed her marriage at the age of 18. Her marital sexual relations are unsatisfactory as coitus rarely culminates in orgasm. In recent years after frustrating unsatisfactory coitus she finally falls asleep and then often dreams of erotic experiences of such intensity that she awakens in orgasm. Shortly after this pleasurable interlude she gradually becomes aware of an increasingly severe cramp in the rectum which follows the pattern of a typical attack of proctalgia fugax.

Because of the unpredictable onset of the spasm and the rapid subsidence of symptoms the only report of endoscopic findings in a patient examined during an attack is that of Bolen (8). He found the rectal mucosa edematous and reddened and the lumen occluded. During observation the edematous mucosa retracted followed by passage of gas, pain relief and subsequent normal appearance of the sigmoid.

It is possible that what Bolen saw was the apex of an intussuscepted sigmoid. Sigmoidorectal intussusception into the rectal ampulla is common. Spasm of the puborectalis muscle constricting the apex of a prolapsed sigmoid may explain the pain in some cases. That this explanation is not too fantastic is indicated by the occurrence of attacks of proctalgia fugax in 35 per cent of 23 patients with demonstrable sigmoidorectal procidentia (9).

As tonic muscle spasm probably is the actual pain producing mechanism its alleviation by a prompt acting antispasmodic medication is indicated. Just as for patients with angina pectoris ampules of amyl nitrite or tablets of glyceryl trinitrite should be provided and be constantly available to patients subject to attacks of proctalgia fugax. Karras and Angelo (7) found that a soluble tablet of nitroglycerin placed under the tongue is simple, convenient, rapid and often effective in aborting or modifying an attack when taken sublingually at the onset of pain. They cited the effectiveness of this simple remedy in 12 patients with proctalgia fugax.

BIZARRE NEUROGENIC DYSCRASIAS

Proctologists constantly see patients who have made the rounds attempting to obtain relief for intolerable discomfort involving the

anus variously described as pressing burning, crawling or aching Exhaustive examinations rarely reveal lesions which would explain the symptom The obviously neurotic patient gives a detailed history of many years of treatment including multiple anal operations, injection treatments, physiotherapy, and numerous local medicaments, all without effect

One must always be on the alert for the woman with symptoms related to the anorectum but with personality traits which place her in the clinical entity described as hysteria (10) In general, these women are friendly, with overtalkativeness a decided characteristic Their history is one of constant sickness with indulgence in excessive medication, they have been excessively hospitalized and operated on, they have many obscure, unrelated complaints Even if definitely indicated anorectal surgical operations rarely result favorably in such patients despite excellent functional and anatomic postoperative results Operations are performed three times more frequently in women with hysteria than in controls, according to a survey by Cohen and his associates (11) The incidence of hemorrhoidectomy in their group was 12 for women with hysteria as against seven for women with organic illness and none for healthy controls

A cursory history pertaining to an individual's general health will readily reveal the psychoneurotic individual who projects his tensions into anal symptoms Surgical procedures for these individuals should be limited to organic lesions which give rise to valid symptoms and which will yield to no other form of treatment save surgery Cryptectomies, papillectomies excision of tags and hemorrhoidectomy for first degree lesions should be avoided in psychoneurotic individuals with anal fixation

REFERENCES

- 1 Duncan G A Painful coccyx Arch Surg 34 1088 1937
- 2a Thiele G H Coccygodynia Am J Surg 79 110 1950
- 2b Thiele G H Coccygodynia and pain in the superior gluteal region JAMA 109 1271 1937
- 3 Granet L Proctalgias and allied perianal dyscrasias Am J Digest Dis 13 330 1946

- 4 Thaysen T E. II Proctalgia fugax Lancet 2 243 1935
- 5 MacLennan A. Rectal crises of non tabetic origin Glasgow M J 88 129 1917
- 6 Blyth W Proctalgia fugax Lancet 2 405 1935
- 7 Karras J H and Angelo G Proctalgia fugax Am J Surg. 82 616 1951
- 8 Bolen H L Spasmodic rectal pain New England J Med 228 564 1943
- 9 Granet E. In discussion on Karras and Angelo (7)
- 10 Purtell J J *et al* Observations on clinical aspects of hysteria JAMA 146 902 1951
- 11 Cohen M E. *et al* Excessive surgery in hysteria JAMA 151 977 1953

Prolapse

THE TERM *prolapse of the rectum* is too general and creates semantic confusion. Prolapse, as applied to the sigmoid and rectum generally describes a pathologic descent of one or more coats of these tubular structures. Buse's simple specific classification of prolapsing lesions based on their pathologic and clinical attributes, includes three types: (1) prolapse of the mucous membrane of the rectum, (2) *procidentia of the rectum* and (3) *sigmoidorectal intussusception*. Bacon too, defines prolapse as referring to the downward displacement of the mucous membrane alone whereas *procidentia* applies to downward displacement of all coats of the bowel.

MUCOSAL PROLAPSE

Primary mucosal prolapse without hemorrhoids is rare in adults but not particularly so in children. Its etiology and its transient nature in young children are discussed in Chapter 5.

Mucosal prolapse (Fig. 104) which causes discomfort because its alkaline mucoid secretion has a traumatizing effect on the perianal skin. The bleeding from erosions of the mucosal surface occasions considerable physical distress and the concomitant constant soiling is disgusting to fastidious individuals. Hemorrhoidal thrombosis, strangulation and eventual slough are frequent complications.

Any treatment short of some type of amputative plastic operation does not effect permanent cure. Sclerosing injections even

when successful afford relief for very short periods only and recurrence is invariable

RECTAL PROCIDENTIA

The term *procidentia* is generally applied to that condition in which all the coats of the rectum including the peritoneum and perhaps the rectosigmoid, are invaginated into the lumen of the



FIG 104 (*left*) —Mucosal prolapse

FIG 105 (*right*) —Rectal procidentia note marked protrusion of anterior rectal wall

rectum and out through the anal aperture Rectal procidentia (Fig 105) is essentially a sliding hernia of the rectum A pouch of peritoneum in the cul-de sac of Douglas (or in the rectovesical pouch) forces its way through a weak area in the pelvic fascia and on to the anterior wall of the rectum The apex thus formed in the rectal wall protrudes into the rectal lumen and descends as it lengthens finally emerging from the anus as a rectal procidentia The hernial sac so formed is then occupied by ileum The posterior rectal wall

which plays a passive role in this mechanism is only slightly protruded Moschcowitz, in 1912, described and demonstrated the hernial mechanism by showing that recurrence after reduction could be prevented by pressing the finger against the anterior rectal wall when pressure was exerted against the posterior wall however prolapse recurred immediately Long duration of the procidentia causes stretching and wide separation of the levator ani muscles and marked relaxation of the pelvic fascia Rectal procidentia occurs predominantly in women in the St Mark's Hospital series the ratio was 5:1, the largest number occurring in the second and third decades

The procidentia, in many cases, has its onset in childhood Left untreated it gradually increases in size until the extensive protrusion completely disables the adult In the aged woman, procidentia is caused by (1) the weakened pelvic fascia resulting from repeated pregnancies (2) inanition, and (3) loss of sphincter muscle tone In most cases, the rectum protrudes after defecation but is usually retained following manual replacement In older patients however the procidentia may persist In all patients with long standing procidentia, the anal sphincter has been so widely stretched that muscle degeneration has destroyed its function as a sphincter This is the reason why relative incontinence frequently persists even after successful surgical repair of the procidentia

TREATMENT

Fifty different methods of treatment for procidentia of the rectum are described in the literature Thirty six methods are commonly used (1) Conservative treatment consists of (1) submucosal injections of sclerosing solutions and (2) the wire splint procedure of Thiersch Bacon describes in detail the various sclerosing techniques for rectal procidentia (2) However the safety and technical advances of modern surgery make the use of sclerosing injections in this condition illogical dangerous and outmoded

In debilitated elderly patients and in patients with incontinence support of the anal ring by the silver wire method of Thiersch (7)

has been revised and advocated by Abel, Gabriel and Dodd all of St Mark's Hospital, London (6) Through two small skin incisions a length of 20 gauge silver wire threaded on a long curved needle is passed around the anus 2 cm from the anal verge so as to encircle the orifice completely The wire is made taut around the assistant's index finger inserted into the anus, tied by twisting and buried in the subcutaneous areolar tissue This mechanical aid to the relaxed anus merely acts as support and its action is similar to the effect of a truss on an inguinal hernia Fecal impaction must be guarded against The wire occasionally breaks in which event it is replaced Gabriel (7) reported gratifying results with the Thiersch operation

Rectosigmoidectomy as modified by Miles is the procedure most often utilized at St Mark's Hospital London for the surgical repair of rectal procidentia Hughes (5) collected and critically evaluated follow up results on 173 patients with rectal procidentia treated by surgery at St Mark's Hospital The best over all long term results followed rectosigmoidectomy Of the 108 patients thus treated the repair was successfully maintained in 43 the procidentia recurred in 65 patients However 21 of the 43 patients with successful surgery were partially incontinent at the anus and four remained with anal stricture

In this country most surgical methods of repair are directed toward eliminating the hernia in the culdesac of Douglas or the rectovesical pouch The pelvic floor is first repaired by approximating the lateral ligaments of the rectum or if possible the levator muscles the redundant sigmoid is then securely sutured to the abdominal parietes in the left pelvic gutter The methods and technique must vary to suit the problem presented by the individual patient The various operations have been evaluated and described by Bacon and his associates (3) in a comprehensive review of the subject This reparative operation affords the best chance for cure as indicated by the fact that there were only four recurrences among 42 patients treated The actual length of the follow up period was not given

SIGMOIDORECTAL INTUSSUSCEPTION

It has been demonstrated sigmoidoscopically that in normal individuals during defecation a small segment of sigmoid prolapses into the ampullary rectum just before the fecal bolus appears at the rectosigmoid (9). Furthermore, it can be noted during routine diagnostic sigmoidoscopy that the sigmoidal mucosa prolapses

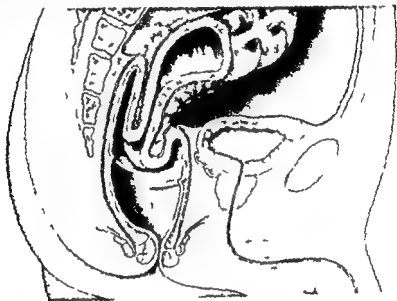


FIG 106—Sigmoidorectal intussusception (Redrawn from Bacon.)

through the rectosigmoid when the patient strains. Slight sigmoidal prolapse, therefore, is physiologic and constitutes a phase of normal defecation.

In many individuals the distal sigmoid is markedly redundant, extremely mobile and possesses a long mesentery. Under certain conditions the expulsive effort of defecation forces a considerable length of redundant mobile sigmoid to prolapse through the rigidly fixed rectosigmoid into the ampullary rectum. This invagination of a segment of sigmoid into the rectum constitutes a true intussusception (Fig. 106). It differs from other types of intussusception in

that the intussusceptum rarely becomes incarcerated or completely obstructed. On the contrary the procidentia occurs intermittently and reduces itself spontaneously.

The anatomic features which facilitate prolapse of the sigmoid are (1) abnormal mobility and redundancy of the distal sigmoid (2) the narrow caliber of the sigmoid as compared with the voluminous lumen of the rectum and (3) the fixation of the recto sigmoid and rectum in the sacral curve by the pelvirectal and sacral fascia. Other factors facilitating prolapse are the force of gravity acting in the normally erect position and the markedly increased intraperitoneal pressure acting in the pelvis during defecation.

Sigmoidorectal intussusception is a common clinical condition which manifests itself by specific symptoms. Although described 70 years ago by Allingham and repeatedly since then in most text books dealing with proctology the syndrome is not well known.

Symptoms resulting from partial intermittent descent of the sigmoid into the rectum vary widely and are often bizarre. The characteristic clinical feature is a sense of unsatisfactory and incomplete evacuation after defecation. The feeling that something remains which must be expelled causes the patient to sit and strain inordinately thus aggravating the tendency to prolapse. As a result of the frequent descent and recession edema, injection and superficial erosion of the mucosa at the apex of the intussusceptum occur often giving rise to glairy and even bloody mucoid discharges. A heavy dragging sensation in the pelvis and in the lumbosacral region may be present together with a dull aching pain referred to the perineum and down the thighs.

When the sigmoid prolapses into the rectum the caliber of lumen of the invaginated bowel is markedly reduced. Patients have found through years of experience that to obtain evacuation through the narrowed lumen the feces must be of semifluid consistency. Laxation with senna, cascara, prune juice, various salines and mineral oil becomes a daily ritual. This constant chemical irritation results eventually in granular, hemorrhagic proctosigmoiditis with tenesmus and frequent ineffectual evacuations. Less frequently patients com-

plain of a form of diarrhea in which, following the passage of a normally formed stool, small segments of feces are repeatedly passed throughout the morning. Postdefecatory proctalgia is common and a history of proctalgia fugax can be elicited in some patients with sigmoidorectal intussusception (9).

Occasionally, in infants and young children colic and straining terminate in bloody and mucoid stools. Proctoscopic examination of such children discloses a granular, hemorrhagic proctosigmoiditis which is limited to the intussusceptum of a sigmoidal procidentia. Schapiro (9a) found prolapse of the sigmoid into the ampulla of the rectum the most common of all types of prolapse in infants and children. Sigmoidorectal intussusception must therefore be considered as a possible source of rectal bleeding in children.

When a patient's history reveals recalcitrant constipation, tenesmus, repeated defections containing blood and mucus, dragging discomfort in the pelvis and perineum, defecatory proctalgia and perhaps proctalgia fugax, sigmoidorectal intussusception as their cause should be considered. Despite its common occurrence the diagnosis of this condition is usually missed because patients are ordinarily examined in the inverted or knee shoulder position. In the inverted patient the force of gravity acting cephalad plus atmospheric pressure entering through the sigmoidoscope replicates the prolapsed sigmoid into its normal anatomic position in the proximal pelvis. Thus, in the inverted position the normal visceral relationship is seen. However, if the patient is examined in Sims' position the procidentia is easily demonstrated. The patient is placed in the recumbent position and the instrument is again passed full length into the sigmoid. Careful air insufflation facilitates this maneuver by dilating the lumen. The sigmoidoscope is then slowly withdrawn and the inflated air allowed to escape by removing the air sealing window. When the instrument reaches the ampullary rectum and the distal aspect of the rectosigmoidal junction is visualized the patient is asked to strain down. The sigmoid can be seen to prolapse through the rectosigmoid usually appearing first at the anterior aspect of the rectum. With further withdrawal of the in-



FIG 107—Sigmoidorectal intussusception segment of sigmoid is invaginated into proximal rectum

strument and continued straining by the patient the apex of the intussusceptum can be seen to descend to the sphincteric rectum in some patients. If digital examination is now performed the apex of the sigmoidal intussusceptum can be palpated as a firm tender knuckle of gut.

A simple technic for its roentgenographic demonstration (Fig 107) has been described (10).

Strangulation or incarceration although uncommon may occur. Monsarrat (10a) reported four cases treated surgically and two additional cases of incarceration of the intussuscepted sigmoid have since been reported (11).

TREATMENT

The constitutional nature of the condition, its long chronicity and the associated hyperirritable colon syndrome all add to the

difficulties of treatment Many patients have exaggerated anxieties regarding their illness, some are in their climacteric others are definitely psychoneurotic.

Anxiety in such patients many of whom have cancerophobia can be relieved by a patient simple explanation of the mechanics of the condition at the conclusion of a very complete examination which must include a barium enema and sigmoidoscopy

The mainstay of treatment is directed to allaying the hyper irritability of the colon and especially spasm in the distal sigmoid (parasympathotonia) Harsh cathartics are banned Anticholinergic drugs of the atropine group are administered in dosage adequate for pharmacologic effect but short of side effects such as dry mouth and mydriasis Most important is the continuous administration of hydrophilic colloids such as psyllium and Karaya, and perhaps the methyl celluloses These hydrophils must be given in frequent and sufficient doses so that the resulting firm bulky, and slippery fecal bolus will dilate and fill the distal sigmoid Such a stool should slip through the rectosigmoidal junction readily and easily to fill the rectal ampulla completely thereby forestalling sigmoidal invagination Mild sedatives essential vitamins and hormones (estrogens androgens thyroid) are adjuvants utilized when indicated by the general medical condition of the patient

Patients are cautioned against straining unduly at stool Should no bowel action result after 48 hours a plain warm water enema taken before retiring is advised This is best administered with the patient either supine or in Sims's position In these positions hydrostatic pressure will mechanically reduce the pluglike intussusceptum and allow the accumulated fecal mass to pass from the sigmoid into the rectum

It should be noted that anorectal inflammatory lesions such as chronic fissures, cryptitis papillitis and pectenosis, are frequently present These lesions must be eradicated surgically in order to eliminate neuromuscular reflex stimuli which so frequently are factors leading to hyperirritability of the colon

Constant reassurance, skillful expectant management of symp

tomatic episodes and considerable patience on the part of the physician as well as of the patient should result in satisfactory therapeutic results

ANAL INCONTINENCE

The severe psychologic distress of the patient with anal incontinence is readily appreciated. Although complete anal incontinence is not frequent, partial incontinence to fluid stools and gas is common following deep perineal tears occurring during parturition or after extensive anorectal operations. The causes of anal incontinence in order of frequency of occurrence are (1) anorectal surgery (high fistula) (2) deep lacerations at parturition (3) direct trauma (accidents, wounds, sodomy) (4) trauma or disease of the spinal cord.

Anal incontinence due to surgical procedures can be prevented if the surgeon rigidly respects fundamental concepts of anatomy and physiology of the anal musculature. The obstetrician should strive to avoid third degree tears by proper prophylactic measures. Should deep lacerations be unavoidable during parturition, they must be promptly aseptically and anatomically repaired. Traumatic wounds involving the sphincters must be treated as the circumstances and the physical findings of the case dictate.

The problem of anal incontinence was thoroughly investigated by Blaisdell (12). His contributions regarding its etiology, physiology and repair are of great value and deserve careful study. Blaisdell emphasizes that not only is muscle incontinence at fault in impaired anal function, but anal deformity resulting from excessive blocks of scar tissue rigidly fixes the anal canal in a position of patency. Various degrees of incontinence result. Mere freeing of the ends of the sphincter from the scar and rejoining them, even if successfully accomplished, rarely increases the mobility of a badly scarred anus. To perform a successful anatomic and functional repair, a revision and reconstruction operation suited to the plastic requirements of the individual case must be designed and accomplished with meticulous care.

Proctology

REFERENCES

- 1 Wright A D In discussion on Prolapse of the Rectum Proc Roy Soc Med 42 1005 1949
- 2 Bacon H E *Anus Rectum Sigmoid Colon* (3d ed Philadelphia J B Lippincott Company 1948)
- 3 Bacon H E Burkett W J and Sauer I Surgical management of rectal procidentia South Surgeon 16 1115 1950
- 4 Gabriel W B *Rectal Surgery* (4th ed London H K Lewis & Co Ltd 1948)
- 5 Hughes E S R *et al* In discussion on Prolapse of the Rectum Proc Roy Soc Med 42 1007 1949
- 6 Dodd H In discussion on Blaisdell P C. Incontinent sphincter ani Am J Surg 79 180 1950
- 7 Gabriel W B Thiersch's operation for anal incontinence and rectal prolapse Am J Surg 86 583 1953
- 8 Goldman C Normal excursion of the sigmoid into the rectum Tr Am Proctol Soc 33 85 1933
- 9 Granet E In discussion on Latras J D and Angelo G Proctalgia fugax Am J Surg 82 616 1951
- 9a. Schapiro S Occurrence of proctologic disorders in infancy and childhood Gastroenterology 15 653 1950
- 10 Granet E Sigmoidorectal intussusception New York State J Med 53 1219 1953
- 10a. Monsarrat A W High third degree prolapse of the rectum Brit J Surg 14 89 1926
- 11 Granet E and Pearl S S Sigmoidorectal intussusception with incarceration Am Pract 3 365 1952
- 12 Blaisdell P C Plastic repair of the incontinent sphincter ani Am J Surg 79 180 1950

REFERENCES

- 1 Wright A D In discussion on Prolapse of the Rectum Proc Roy Soc Med 42 1005 1949
- 2 Bacon H E *Anus Rectum Sigmoid Colon* (3d ed Philadelphia J B Lippincott Company 1948)
- 3 Bacon H E Burkett W J and Sauer I Surgical management of rectal procidentia South Surgeon 16 1115 1950
- 4 Gabriel W B *Rectal Surgery* (4th ed London H K. Lewis & Co Ltd 1948)
- 5 Hughes E S R *et al* In discussion on Prolapse of the Rectum Proc Roy Soc Med 42 1007 1949
- 6 Dodd H In discussion on Blaisdell P C. Incontinent sphincter ani Am J Surg 79 180 1950
- 7 Gabriel W B Thiersch's operation for anal incontinence and rectal prolapse Am J Surg. 86 583 1953
- 8 Goldman C. Normal excursion of the sigmoid into the rectum Tr Am Proctol Soc 33 85 1933
- 9 Granet E. In discussion on Karras J D and Angelo G Proctalgia fugax Am J Surg 82 616 1951
- 9a Schapiro S Occurrence of proctologic disorders in infancy and childhood Gastroenterology 15 653 1950
- 10 Granet E Sigmoidorectal intussusception New York State J Med 53 1219 1953
- 10a Monsarrat K. W High third degree prolapse of the rectum Brit J Surg 14 89 1926
- 11 Granet E and Pearl S S Sigmoidorectal intussusception with incarceration Am Pract 3 365 1952
- 12 Blaisdell P C. Plastic repair of the incontinent sphincter ani Am J Surg. 79 180 1950

Diverticula of the Colon

SINCE 1914 when Carmen first described its roentgenographic features several hundred reports on diverticular disease of the colon have appeared. Various opinions have been expressed in the literature regarding its incidence, sex preponderance, relationship to habitus, complications, and treatment. It is agreed generally that diverticula are rarely seen before the age of 30 and that the incidence increases directly with age.

Edwards (1) reported an over all incidence of 12 per cent. Below age 35 the incidence was 0.6 per cent; above this age the incidence averaged 16 per cent and rose to 37.5 per cent at age 75 or over. Among his private patients examined by barium enema Bockus found colonic diverticula in 8 per cent. In a comparable series of mine of 447 private patients colonic diverticula were diagnosed by barium enema in 14 per cent; only three patients of 113 in this series below the age of 40 had diverticula.

Most reported figures indicate that diverticula occur slightly more frequently in the male than in the female and that diverticulitis and other inflammatory complications predominate in the male. Ochsner and Bergen (2) found male preponderance in the ratio of 1.25:1 in diverticulosis as against 2.75:1 for diverticulitis. Body habitus has little significance in this disease. The sigmoid is the site of predilection in colonic diverticula, being involved solely or in part in approximately 85 per cent of reported cases.

The exact cause of the formation of diverticula is not known.

REFERENCES

- 1 Wright A B In discussion on Prolapse of the Rectum Proc Roy Soc Med 42 1005 1949
- 2 Bacon H E *Anus Rectum Sigmoid Colon* (3d ed Philadelphia J B. Lippincott Company 1948)
- 3 Bacon H E, Burkett, W J and Sauer J Surgical management of rectal procidentia South Surgeon 16 1115 1950
- 4 Gabriel W B *Rectal Surgery* (4th ed London H K. Lewis & Co. Ltd 1948)
- 5 Hughes E S R. *et al* In discussion on Prolapse of the Rectum Proc. Roy Soc. Med 42 1007 1949
- 6 Dodd H In discussion on Blaisdell P C. Incontinent sphincter ani Am. J Surg 79 180 1950
- 7 Gabriel W B Thiersch's operation for anal incontinence and rectal prolapse Am J Surg 86 583 1953
- 8 Goldman C. Normal excursion of the sigmoid into the rectum Tr Am Proctol Soc 33 85 1933
- 9 Granet E. In discussion on Karras J D and Angelo G Proctalgia fugax Am J Surg. 82 616 1951
- 9a. Schapiro S Occurrence of proctologic disorders in infancy and childhood Gastroenterology 15 653 1950
- 10 Granet E. Sigmoidorectal intussusception New York State J Med 53 1219 1953
- 10a. Monsarrat A W High third degree prolapse of the rectum Brit J Surg. 14 89 1926
- 11 Granet E and Pearl S S Sigmoidorectal intussusception with incarceration Am Pract 3 365 1952
- 12 Blaisdell P C. Plastic repair of the incontinent sphincter ani Am J Surg 79 180 1950

Diverticula of the Colon

SINCE 1914 when Carmen first described its roentgenographic features several hundred reports on diverticular disease of the colon have appeared. Various opinions have been expressed in the literature regarding its incidence, sex preponderance, relationship to habitus, complications, and treatment. It is agreed generally that diverticula are rarely seen before the age of 30 and that the incidence increases directly with age.

Edwards (1) reported an over all incidence of 12 per cent. Below age 35 the incidence was 0.6 per cent; above this age the incidence averaged 16 per cent and rose to 37.5 per cent at age 75 or over. Among his private patients examined by barium enema Bockus found colonic diverticula in 8 per cent. In a comparable series of mine of 447 private patients colonic diverticula were diagnosed by barium enema in 14 per cent; only three patients of 113 in this series below the age of 40 had diverticula.

Most reported figures indicate that diverticula occur slightly more frequently in the male than in the female and that diverticulitis and other inflammatory complications predominate in the male. Ochsner and Bagen (2) found male preponderance in the ratio of 1.25:1 in diverticulosis as against 2.75:1 for diverticulitis. Body habitus has little significance in this disease. The sigmoid is the site of predilection in colonic diverticula, being involved solely or in part in approximately 85 per cent of reported cases.

The exact cause of the formation of diverticula is not known.

nor is there agreement as to the mechanism of their formation. A *diverticulum* is a hernia through the walls of the bowel as with all hernias increased pressure or strain on weakened or aging fascial and muscle layers causes the latter to give way—in this case to an out pouching of the colonic *mucosa* (Fig 108) The route taken by the mucosal sac is probably through the area where the integrity of



FIG 108—Pathogenesis of colonic diverticula. *a* congenital gap in circular muscle *b* mucosal hernia (diverticulum) (Figs 108 and 109 from O V Lloyd Davies Proc. Roy Soc Med 46 407 1953)

intestinal muscle is defective. Such areas are present at the points where the blood vessels penetrate the longitudinal and circular musculature to make their way from the serosa to the submucosa. Edwards believes that mucosal out pouching takes place as a result of constant intraluminal pressure. On the basis of his extensive surgical observations Erdman believes that diverticula more frequently occur on the convex and lateral aspects of the sigmoid at a considerable distance from the vessels and frequently emerge under the appendices epiploicae.

As diverticula are passive mucosal layer pouches without expulsive musculature between the submucosa and its serosal covering a chain of events may occur which leads to inflammatory lesions. Fecaliths form in the sac and erode the mucosal lining this is followed by inflammatory lesions in the submucosa which spread by contiguity to the adjacent intestinal wall and result in widespread sigmoiditis. Many complications are caused by inflammation starting in the diverticula and they cause difficult therapeutic problems.

SYMPTOMS AND DIAGNOSIS

SYMPTOMS—Diverticulosis is a silent lesion and is usually found unexpectedly during diagnostic roentgenographic investigation of the gastrointestinal tract. Symptoms are a consequence of infection arising in the diverticula. Diverticulitis ensues in approximately 20 per cent of patients with diverticula. Disturbed bowel function is a result of the associated inflammation.

Bowel irritability is manifested by hypermotility and diarrhea or it may give rise to spasm and cause constipation. Pain may result from spasm in early lesions from the inflammatory process in the sigmoid or as a consequence of extension of the inflammation to adjacent organs. Intestinal obstruction may occur as a consequence of inflammatory sigmoiditis or of a peritoneal abscess. Melena and gross hemorrhage is uncommon in diverticulitis but when it occurs causes great concern. In a patient reported on by Donald (3) temporary ileostomy was necessary. In severe cases with complications requiring hospitalization Patterson (4) reported melena in 16 per cent of 434 patients. The bleeding may stem from some cause other than the diverticula. In all cases of diverticula with melena the bowel should be carefully investigated for carcinoma or polyps. Severe abdominal pain, local tenderness, distention and vomiting are signs of an acute condition requiring immediate surgical aid.

DIAGNOSIS—Roentgenography is the most important diagnostic aid. In the presence of sigmoidal diverticulosis narrowing of the lumen and possible obstruction arouse keen suspicion that carcinoma may also be present. In 25 per cent of the cases of divertic

ulitis reported by Pemberton *et al* (5), the possible presence of carcinoma could not be satisfactorily excluded by x ray study alone

The sigmoidoscope may help in the search for diverticula of the sigmoid. Actual visualization of the orifice of the diverticulum is very rare. Buie and his co workers at the Mayo Clinic have established certain criteria which in their opinion indicate the presence



FIG. 109—Sacculations in diverticulosis.

of sigmoidal diverticula. Buie and Jackman (6) made an endoscopic diagnosis of diverticula of the sigmoid in 160 of 242 proved cases. The criteria are

1. Sacculation of the sigmoid (Fig. 109). These shallow pouches separated by diaphragmatic elevations across the lumen of the bowel are seen best when the lumen is distended with air. Of the 160 cases, 72 showed sigmoidal sacculations.

2. Visualization of the orifice of a diverticulum. This was seen in 35 of the 160 cases.

3 Indirect evidence of diverticula consisting of (*a*) relative immobility of the sigmoid (*b*) sharp angulation of the bowel lumen, and (*c*) narrowing of the bowel lumen and crowding and exaggeration of mucosal folds. Such evidence was found in 53 cases.

Buie frankly concedes that these indirect signs are frequently due to extraneous causes such as pelvic inflammatory or neoplastic disease, redundancy of the sigmoid and short mesentery.

In many of my cases of diverticula seen by x rays to be low in the sigmoid endoscopy showed a narrowed lumen edema crowding of the rugae and also immobility of the sigmoid. I have often felt that if the folds could be separated an orifice leading into a diverticulum could be seen but careful use of air insufflation did not separate the mucosal folds. Air insufflation must be used cautiously in cases of suspected diverticular disease because very little intraluminal air pressure is required to perforate the thin walled sacs. Advance of the instrument is blocked in most cases by angulation and fixation of the bowel extreme care is essential since a fixed rigid intestine is easily perforated by the metal instrument. The reliability of the sacculations described by Buie as a diagnostic sign of diverticula has been confirmed (7, 8).

COMPLICATIONS

The consensus of reports indicates that diverticulitis and sigmoiditis occur in 15 to 20 per cent of patients with diverticulosis. The symptom complex of acute inflammation in sigmoidal diverticula has been referred to as left sided appendicitis. The inflammation may subside or may progress and finally cause perforation. Peritonitis is the result of perforation frequently a walled off abscess develops which may form a palpable left lower quadrant mass or which may be felt as a pelvic mass on digital rectal examination. Adhesion to an adjacent viscus may result in perforation and the development of internal fistulas—sigmoidovesical and sigmoidenteric. Drainage of an abscess starting in diverticulitis may result in sigmoidocutaneous fistulas (9). Intestinal obstruction partial or

complete may occur as the result of the sigmoiditis or of an obstructing abscess Flynn (10) reviewed the findings in 110 surgically treated complications of diverticulitis (Table 5)

The problem of differentiation of sigmoidal diverticulosis and carcinoma has been mentioned (5) Even at operation with the sigmoid visualized and palpable accurate diagnosis is frequently

TABLE 5—INCIDENCE OF COMPLICATIONS IN 100 CASES OF DIVERTICULOSIS (10)

COMPLICATION	No. of Cases	In Carc.
Perforation (92 cases 92%) resulting in		
Mass	74	67
Abdominal	35	30
Pelvic	19	17
General peritonitis	18	16
Intestinal obstruction (50 cases 44%)		
Partial	43	40
Complete	7	1
Fistula (29 cases 25%)		
Sigmoidocutaneous	19	17
Sigmoidovesical	6	3
Sigmoidoenteric	4	3

impossible The final word must come from the microscope and the pathologist

Carcinoma does occur in sigmoids that contain diverticula but the relation of diverticulitis to carcinoma is apparently incidental Morton (11) reported in detail on 18 cases of coexisting diverticulitis and carcinoma of the sigmoid treated surgically during a 20 year period at the University of Rochester Hospitals a similar number of complicated cases of diverticulitis were proved to be free of cancer only after radical resection and microscopic examination

An interesting and probably not infrequent complication has been described by Cohen (12) as a dissecting intramural diverticulitis In the two cases reported the lesion consisted of a herniation of the mucosa which burrowed into the wall of the intestine and extended longitudinally along the wall instead of penetrating to the serosa

TREATMENT

Donald (3) compared the management of diverticulitis to that of duodenal ulcer in that surgery is reserved primarily for the complications which occur in 10 to 20 per cent of the patients. Chronic uncomplicated diverticulitis should be managed medically with a bland diet and supportive expectant treatment. For patients who have surgical exploration Donald advised medical therapy if uncomplicated acute diverticulitis is found.

For complicated diverticulitis the procedure varies according to the findings and the experience of the surgeon. All surgeons agree that success in treatment of diverticulitis and its complications calls for individualization and great surgical ingenuity. The various aspects of surgical treatment have been clearly discussed by Donald.

REFERENCES

- 1 Edwards H C. *Diverticula and Diverticulosis of the Intestine* (Bristol J Wright & Sons Ltd 1939)
- 2 Ochsner H C. and Bagen J A. Diverticulosis of the large intestine. *Ann Int Med* 9:282 1935
- 3 Donald J M. The surgical management of diverticulitis of the colon. *Ann Surg.* 133:708 1951
- 4 Patterson C O. Diverticulitis of the colon. *Gastroenterology* 18:201 1951
- 5 Pemberton J deJ. *et al*. Surgical management of diverticulitis of the sigmoid colon. *Surg. Gynec. & Obst.* 83:523 1947
- 6 Buie L A. and Jackman R J. Diverticula of the colon. *JAMA* 121:1144 1945
- 7 Young V T. Results of routine sigmoidoscopy. *Rev Gastroenterol* 18:283 1951
- 8 Steele H H. and Brown C H. Analysis of 1500 routine proctosigmoidoscopic examinations. *Gastroenterology* 12:419 1949
- 9 Mayfield L H. and Waugh J M. Sigmoidocutaneous fistulae resulting from diverticulitis of the sigmoid-colon. *Ann Surg.* 129:198 1949
- 10 Flynn J E. Surgical aspects of diverticulitis of the sigmoid. *Rev Gastroenterol* 17:577 1950
- 11 Morton J J. Diverticulitis and carcinoma of the sigmoid. *Surgery* 52:765 1952
- 12 Cohen S E. Dissecting (intramural) diverticulitis. *New York State J Med* 41:2661 1949

complete, may occur as the result of the sigmoiditis or of an obstructing abscess Flynn (10) reviewed the findings in 110 surgically treated complications of diverticulitis (Table 5)

The problem of differentiation of sigmoidal diverticulosis and carcinoma has been mentioned (5) Even at operation with the sigmoid visualized and palpable, accurate diagnosis is frequently

TABLE 5—INCIDENCE OF COMPLICATIONS IN 100 CASES OF DIVERTICULOSIS (10)

COMPLICATION	No. of CASES	PERCENT
Perforation (92 cases 92%) resulting in		
Mass	74	67
Abdominal	55	50
Pelvic	19	17
General peritonitis	18	16
Intestinal obstruction (50 cases 44%)		
Partial	45	40
Complete	5	4
Fistula (29 cases 25%)		
Sigmoidocutaneous	19	17
Sigmoidovesical	6	5
Sigmoidoenteric	4	3

impossible The final word must come from the microscope and the pathologist

Carcinoma does occur in sigmoids that contain diverticula, but the relation of diverticulitis to carcinoma is apparently incidental Morton (11) reported in detail on 18 cases of coexisting diverticulitis and carcinoma of the sigmoid treated surgically during a 20 year period at the University of Rochester Hospitals a similar number of complicated cases of diverticulitis were proved to be free of cancer only after radical resection and microscopic examination

An interesting and probably not infrequent complication has been described by Cohen (12) as a dissecting intramural diverticulitis In the two cases reported the lesion consisted of a herniation of the mucosa which burrowed into the wall of the intestine and extended longitudinally along the wall instead of penetrating to the serosa

TREATMENT

Donald (3) compared the management of diverticulitis to that of duodenal ulcer in that surgery is reserved primarily for the complications which occur in 10 to 20 per cent of the patients. Chronic uncomplicated diverticulitis should be managed medically with a bland diet and supportive, expectant treatment. For patients who have surgical exploration Donald advised medical therapy if uncomplicated acute diverticulitis is found.

For complicated diverticulitis the procedure varies according to the findings and the experience of the surgeon. All surgeons agree that success in treatment of diverticulitis and its complications calls for individualization and great surgical ingenuity. The various aspects of surgical treatment have been clearly discussed by Donald.

REFERENCES

- 1 Edwards H C. *Diverticula and Diverticulosis of the Intestine* (Bristol: J Wright & Sons Ltd, 1939).
- 2 Ochsner H C and Bagen J A. Diverticulosis of the large intestine. *Ann Int Med* 9: 282, 1935.
- 3 Donald J M. The surgical management of diverticulitis of the colon. *Ann Surg.* 133: 708, 1951.
- 4 Patterson C O. Diverticulitis of the colon. *Gastroenterology* 18: 201, 1951.
- 5 Pemberton J deJ *et al*. Surgical management of diverticulitis of the sigmoid colon. *Surg. Gynec & Obst* 85: 523, 1947.
- 6 Buie L A and Jackman R J. Diverticula of the colon. *JAMA* 121: 1144, 1945.
- 7 Young V T. Results of routine sigmoidoscopy. *Rev Gastroenterol* 18: 283, 1951.
- 8 Steele H H and Brown C H. Analysis of 1500 routine proctosigmoidoscopic examinations. *Gastroenterology* 12: 419, 1949.
- 9 Mayfield L H and Waugh J M. Sigmoidocutaneous fistulae resulting from diverticulitis of the sigmoid-colon. *Ann Surg.* 129: 198, 1949.
- 10 Flynn J E. Surgical aspects of diverticulitis of the sigmoid. *Rev Gastroenterol* 17: 577, 1950.
- 11 Morton J J. Diverticulitis and carcinoma of the sigmoid. *Surgery* 32: 765, 1952.
- 12 Cohen S E. Dissecting (intramural) diverticulitis. *New York State J Med* 41: 2661, 1949.

CHAPTER SEVENTEEN

Pilonidal Disease

THE INCLUSIVE TERM pilonidal disease should be used to describe all clinical forms of the disease including pilonidal sinus, pilonidal cyst and cyst and sinus combined. The lesion may be entirely quiescent or it may be actively inflamed and take the form of an acute abscess or subacute or chronic sinocystic disease. It is because of its pathologic and clinical vagaries that contemporary concepts of its etiology and its surgical management are still controversial.

Although pilonidal disease is a significant clinical entity in our population cases requiring surgical care were uncommon before World War II. McKirdie (1) found the ratio of hospital admissions for pilonidal disease to all hospital admissions to be 1:1,500. Careful physical examination will disclose an actual quiescent pilonidal sinus in 3 to 4 per cent of all adults. An astounding rise in the incidence of active pilonidal disease in military personnel appeared during World War II and since then. The number of man days lost to this disease in the Army during this period is estimated at about 3,470,000 (2). In the Navy, between 1944 and 1951, 530,701 man days were lost because of pilonidal disease (3). In 1913 and 1944 at the U.S. Naval Hospital, St. Albans, N.Y., 7 per cent of all operations on the general surgical service were for the definitive treatment of inflammatory pilonidal disease. The probable explanation for the activation of this ordinarily quiescent disease by the war is the impact of unaccustomed trauma, heat, sweat and dirt.

associated with certain phases of military life. Another factor of importance is the alacrity with which military patients accept the security of hospitalization and definitive operation for their draining sinus which they probably would tolerate indefinitely as civilians.

Pilonidal disease rarely occurs in Negroes and in other non-Caucasian races. There is no adequate explanation for this apparent racial selectivity.

ETIOLOGY

A clear understanding of the fundamental factors which play a role in the pilonidal lesion is essential in planning its treatment. The pilonidal cyst or sinus has hitherto been considered an infected embryologic remnant. The two main theories of the origin of this remnant are: (1) the sinus arises from the ectoderm by imperfect separation of the caudal end of the neural tube; (2) it is a sequestration dermoid formed during fusion of the ectoderm growing in toward the midline. By sequestration dermoid is meant a cyst formed along the line of cleavage in the embryo. There is proof that both of these mechanisms give rise to dermoids in this region.

Patey and Scarf in England, King in Melbourne, and Smith in this country challenge the view that all or even most pilonidal cysts and sinuses are of embryonic origin. They believe that most examples are acquired infected lesions probably originating from puncture of the skin by a hair.

NEUROGENIC RESTS—Gage (4) and more recently Kooistra (5) reviewed and restudied the embryology of the caudal end of the human embryo. It has been shown conclusively that the medullary canal, the anlage of the central nervous system, does not develop uniformly. In an 11 cm. long embryo the extreme tail portion of the medullary canal can be seen to have been closed off to form a simple epithelial sac which assumes the character of a cystlike space. During the second half of fetal life this cystlike structure undergoes gradual atrophy, becoming not only smaller but also tending to be replaced by connective tissue. Gage pointed out that the neural canal is completely formed before the appearance of the

most primitive of the appendages of the epidermis i.e., hair shafts, sebaceous glands, and sweat glands. Therefore, in sections of pilonidal cysts and sinuses these appendages of the epidermis should be completely lacking.

ECTODERMAL INVAGINATION—The theory that pilonidal sinus results from a form of embryonic invagination of the ectoderm was first suggested by Lannelongue in 1886 and later re-investigated by Fox (6) who studied serial sections through the caudal region of seven human embryos varying in age from 14 to 30 weeks. Fox demonstrated that grooves and creases appeared in the skin over the coccyx in the 3½ month fetus. Microscopic examination of this 99 mm embryo showed active invaginating ectoderm. In older embryos (just before viability) the invagination increased in depth and impinged directly on the coccyx. The skin appendages especially the hair follicles, are particularly abundant in the region of invagination. After viability, only slight evidence of invaginated ectoderm (pilonidal cleft) remains and is probably due to a recession of the invaginating process. It is assumed that pilonidal sinus occurs when the process of recession does not take place. Multiple congenital orifices varying in size from pinpoint to several millimeters are commonly seen in pilonidal disease. These according to Fox are derived from multiple invaginations.

TRAUMATIC INFECTION—Patey and Scarf (7), in 1916 reported the case of a barber with a discharging sinus tract in the interdigital cleft between the fourth and the fifth finger of the right hand. This apparently resulted from penetration of the skin by cut hair from a client's head. Histologic examination of a pea sized nodule excised en bloc showed a sinus lined with squamous epithelium which led into a cavity lined with granulation tissue and containing hair and debris in its wall. These authors believed that this case fulfilled all morphologic criteria consistent with the diagnosis of pilonidal sinus and cyst. Since then 10 additional cases of interdigital pilonidal sinus have been found in the literature.

In 1948 Smith (8) reported four cases of sinuses and cysts occurring in the anterior perianal region. The presumptive diagnosis

was anal fistula and they were excised. When opened they were found to contain hair and histologic examination revealed morphology similar to that seen in pilonidal sinuses. In none of these patients was the sinus opening situated in the perineal raphe (midline) nor was the tract directed toward the anus. The eccentric location of the sinus disposes of the theory of ectodermic invagination as applied to anterior perineal cysts. In these cases the neurogenic theory is untenable since no embryonic vestige of the neural canal is normally located in the anterior perineum.

According to King (9) the main features which conform with and support the traumatic infectious hypothesis and are not explained easily by any congenital etiologic theory are (1) the maximum incidence in the second and third decades (2) the presence of multiple lesions (3) the occurrence of lesions in locations other than the midline and (4) the presence of hair as a foreign body and not as a local formation.

HAIR IN PILONIDAL LESIONS—The presence of hair in sinuses and cysts is typical of the lesion. In 1880 Hodges (10) described the hair as present in nests and as being short without bulbs but sometimes a root may be found but only at the peripheral end. King said: "It is somewhat astonishing therefore to find that in almost all subsequent writings the hairs have been considered to arise locally and the extraneous origin of them has been entirely overlooked."

In the series of cases at St. Albans Naval Hospital (11) hair was not found in any sacrococcygeal cyst which had no demonstrable sinus leading to the skin surface. In our opinion the subcutaneous cysts are neurogenic in origin. No evidence was found in our cases of hair follicles in the sections through the deep portion of sinuses and cysts of excised specimens. Furthermore none of the photomicrographs accompanying the numerous articles that I have seen clearly show hair follicles in the cyst wall; what is visible in the illustrations are cross sections of hair shafts. Follicles have been demonstrated only occasionally in sections taken close to the sinus orifice on the skin surface. I am convinced that hair found in

sinuses and cysts of extraneous origin. Individual hairs from the skin surface work their way into the sinus and finally into the cyst. Eventually, through years of accumulation and by constant molding from external mechanical pressure they become compressed and kneaded into so-called hair nests.

PATHOLOGY

The sinuses, infected cysts, and abscesses of pilonidal disease differ little from chronic fistulas and abscesses around the ano-rectum. Squamous epithelium is found lining the sinuses if they are quiescent and of long standing. This lining epithelium is destroyed progressively with increasing chronic infection. Surrounding the sinus or cyst is a fibrous tissue capsule the thickness of which increases with the duration of the inflammatory stage of the disease (Fig. 110). In the excision operation for chronic pilonidal disease the presence of this thick capsule aids in the dissection of the cyst or sinus from the surrounding subcutaneous areolar tissue. Acute abscesses contain a considerable amount of pus, in addition there is associated cellulitis of the surrounding areolar tissue as evidenced by swelling and a hot, reddened skin.

CLINICAL FEATURES

In the literature there is a general disregard for the classification of the clinical forms of this disease. The general tendency is to consider pilonidal disease as acute or chronic and no attempt is made to distinguish between the various anatomic forms of the lesion. This has resulted in the fallacious attempt to treat entirely different types of the disease by one standard method with consequent failure as indicated by the high percentage of recurrences.

In the 20 months during which I served in St. Albans Naval Hospital 319 patients underwent surgery for the definitive eradication of pilonidal disease. The clinical findings in these patients may be reviewed briefly. In primary cases, i.e. those without previous definitive operation, the commonest lesion was the single or multiple infected sinus (35 per cent). Acute abscesses were next (29 per

cent) followed closely by sinuses and cysts combined (28 per cent) Infected subcutaneous cysts alone without a demonstrable sinus leading to the skin were seen in 21 patients In these the specimen when opened after excision revealed chronic inflammatory granulation tissue occasionally mucopus but never intraluminal hair

Clinical symptoms varied with the specific pathologic forms of



FIG 110 —Cross section of chronic pilonidal sinus note hair and debris in lumen intact squamous epithelial lining (right inferior) and thick fibrous capsule surrounding sinus tract (From M Gage Arch Surg 31 175 1935)

the disease Quiescent sinuses did not give rise to symptoms and required no treatment Infected sinuses were tender and discharged varying quantities of mucopus Solitary cysts without sinuses caused symptoms only when inflamed when quiescent they were merely palpable and not tender Abscesses caused pain swelling and fever They ruptured spontaneously or emptied to the skin surface through their associated sinuses These spontaneous evacuations were fol

sinuses and cysts is of extraneous origin. Individual hairs from the skin surface work their way into the sinus and finally into the cyst. Eventually, through years of accumulation and by constant molding from external mechanical pressure they become compressed and kneaded into so-called hair nests.

PATHOLOGY

The sinuses, infected cysts, and abscesses of pilonidal disease differ little from chronic fistulas and abscesses around the anorectum. Squamous epithelium is found lining the sinuses if they are quiescent and of long standing. This lining epithelium is destroyed progressively with increasing chronic infection. Surrounding the sinus or cyst is a fibrous tissue capsule, the thickness of which increases with the duration of the inflammatory stage of the disease (Fig. 110). In the excision operation for chronic pilonidal disease the presence of this thick capsule aids in the dissection of the cyst or sinus from the surrounding subcutaneous areolar tissue. Acute abscesses contain a considerable amount of pus; in addition there is associated cellulitis of the surrounding areolar tissue, as evidenced by swelling and a hot reddened skin.

CLINICAL FEATURES

In the literature there is a general disregard for the classification of the clinical forms of this disease. The general tendency is to consider pilonidal disease as acute or chronic, and no attempt is made to distinguish between the various anatomic forms of the lesion. This has resulted in the fallacious attempt to treat entirely different types of the disease by one standard method with consequent failure as indicated by the high percentage of recurrences.

In the 20 months during which I served in St. Albans Naval Hospital 319 patients underwent surgery for the definitive eradication of pilonidal disease. The clinical findings in these patients may be reviewed briefly. In primary cases, i.e., those without previous definitive operation, the commonest lesion was the single or multiple infected sinus (35 per cent). Acute abscesses were next (29 per

cent) followed closely by sinuses and cysts combined (28 per cent). Infected subcutaneous cysts alone without a demonstrable sinus leading to the skin were seen in 21 patients. In these the specimen when opened after excision revealed chronic inflammatory granulation tissue occasionally mucopus but never intraluminal hair.

Clinical symptoms varied with the specific pathologic forms of



FIG 110—Cross section of chronic pilonidal sinus note hair and debris in lumen intact squamous epithelial lining (right inferior) and thick fibrous capsule surrounding sinus tract. (From M Gage Arch Surg 31 175 1935)

the disease. Quiescent sinuses did not give rise to symptoms and required no treatment. Infected sinuses were tender and discharged varying quantities of mucopus. Solitary cysts without sinuses caused symptoms only when inflamed; when quiescent they were merely palpable and not tender. Abscesses caused pain, swelling, and fever. They ruptured spontaneously or emptied to the skin surface through their associated sinuses. These spontaneous evacuations were fol-

lowed by rapid subsidence to the subacute or chronic phase. Repeated episodes of acute abscess formation were common in our patients.

It would take us too far afield at this point to discuss in detail the recurrent cases in our series of 319 patients. They numbered 78 (25 per cent) and recurred after purported definitive treatment, 43 of them followed attempts at definitive primary closure elsewhere and 16 did not heal or broke down following previous operation by open packing or by partial closure.

TREATMENT

It is generally accepted that primary closure of wounds following excision of chronically infected pilonidal cysts and sinuses when successful heals in the shortest time and affords the most satisfactory scar. However, many cases that are ill suited for primary closure have nevertheless been so treated, on the assumption that if the wound became infected and broke down sutures could be removed and the resultant gaping wound be allowed to heal in by granulation. This was not so in our cases. Following removal of sutures the grossly infected wound healed slowly, as is true of all grossly infected granulation tissue, so that eventual epithelization was markedly delayed.

Some surgeons attempt primary closure of infected wounds after a prolonged period of preoperative treatment consisting of bed rest, wet dressings and antibiotic medication. The prolonged hospitalization nullifies the advantages of primary closure insofar as the time saving factor is concerned. Failure of primary closure in many of our cases was due to neglect of physiologic principles essential for proper wound healing. Among these were (1) attempted closure of grossly infected wounds too soon after incision and drainage of an acute suppurative pilonidal abscess, (2) attempted closure of defects so wide that suture lines cut through or gave way to excessive tension resulting in dead space and eventual infection, (3) use of chromic catgut as an approximating suture. An excessive number of wounds early in our series became infected despite the fact that

the sutured case was particularly favorable for primary closure. The breakdown in the wound was possibly due to excessive tissue reaction to the chromic catgut. The tissue irritation of the chromic catgut, together with the action of septic organisms always present in these wounds apparently overwhelmed the process of normal healing and resulted in gross infection. In our later cases catgut was discarded as suture material.

PRIMARY CLOSURE—For pilonidal disease primary closure should be used only in carefully selected cases in which (1) months have elapsed since any acute flare up so that infection is limited to the lumen of the tract, (2) the wound is of limited width after excision of the lesion and (3) complete cooperation from the patient in meticulous postoperative care may be expected. The technic of primary closure which gave satisfactory results in properly selected cases was that originally described by Ferguson (14) in 1935. It is based on sound physiologic principles of wound healing and the technic is relatively simple. I have modified it only by utilizing currently available antibiotics as adjuvants.

After preoperative sedation and precise skin preparation local field block with 1 per cent procaine solution containing 8 minims of 1:1,000 solution of epinephrine to each 100 cc is instituted in the clean and aseptic field surrounding the sinus tract. Immediate anesthesia and a relatively avascular operative field result. The skin adjacent to and below the distal sinus opening is incised and dissected free. A towel clip through the distal sinus tract held in the operator's left hand and small sharp retractors on the skin margins held by the assistant serve to place the tissues under tension so that dissection of the lesion is facilitated. Chronicity of the lesion produces marked fibrosis around the cysts and sinuses so that with careful scalpel dissection the lesion can be shelled out while removing only a minimum of surrounding normal skin and areolar tissue (Fig. 111). It is essential to keep the level of the dissection as superficial as possible so as to preserve the all important postsacral fascia. However, complete removal of the sinus tract must be assured. Lateral extensions are dissected out when found. additional procaine

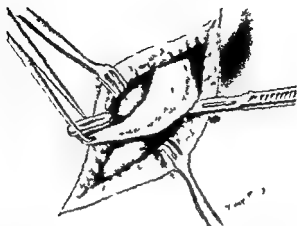


FIG 111—Excision of pilonidal sinus and cyst by dissection

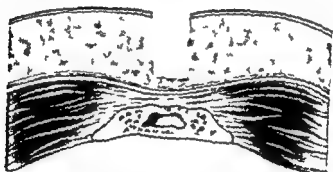


FIG 112—Type of wound considered favorable for primary closure post-sacral fascia is intact and lateral walls do not gape

solution being infiltrated when necessary. Bleeding is usually minimal, due to the use of epinephrine when encountered is controlled by fine hemostats.

The wound is considered favorable for primary closure only if the lateral walls can be approximated without undue tension and only if the post-sacral fascia remains intact (Fig. 112). If these criteria are present hemostats are removed residual bleeders if any,

are caught and tied with very fine plain catgut and the wound is then thoroughly irrigated with sterile saline solution. Adhesive retracting straps are removed and the skin is again prepared and redraped. With a full curved cervix needle, sutures of very fine alloy steel wire (no 35) are placed at short intervals starting at the distal end of the wound. The suture incorporates the deep areolar tissue on both sides as well as a wide portion of the postsacral fascia (Fig 113). It is technically easier to place all sutures before tying. Extremely fine wire is used for two reasons: (1) it causes practically no tissue reaction and (2) its low tensile strength insures against excess tension in the wound. If the suture will not hold tension is considered excessive and primary closure in such a wound is thereby precluded. In deep defects a second superficial layer of no 35 wire sutures is used. All knots must be square tied three times and cut on the knot to avoid sharp ends. Finally through and through vertical mattress sutures of no 28 wire are passed on large curved cutting needles starting 3 cm from the near skin edge. The near end is then threaded on a fine cutting needle and the skin edges are approximated. The tension sutures are tied firmly over gauze rolls (Fig 114). A dry dressing firmly held by wide adhesive straps placed across the buttocks completes the procedure.

The patient may be partially ambulatory after the third postoperative day but sitting is forbidden. The primary dressing is retained until the tenth postoperative day. Tension sutures are then removed and an alcohol dressing is applied and firmly fixed with wide adhesive straps. Dressings are discontinued after the fourteenth postoperative day. To avoid pressure and tension on the fresh wound the patient is cautioned to sit only on the lateral portion of the thigh and buttock with the opposite leg crossed over the knee during convalescence and for some time thereafter. As prophylaxis 600 000 units of procaine penicillin and 1 Gm of streptomycin are administered the day before operation and daily through the fifth postoperative day.

Recurrence is often attributed to the fact that some portion of the sinus tract remained in the wound due to incomplete removal

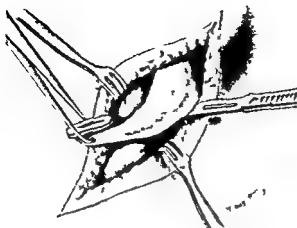


FIG 111 —Excision of pilonidal sinus and cyst by dissection

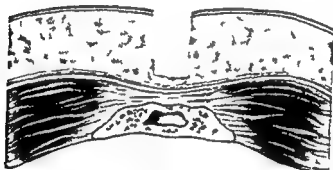


FIG 112 —Type of wound considered favorable for primary closure post-sacral fascia is intact and lateral walls do not gape

solution being infiltrated when necessary. Bleeding is usually minimal due to the use of epinephrine. When encountered it is controlled by fine hemostats.

The wound is considered favorable for primary closure only if the lateral walls can be approximated without undue tension and only if the postsacral fascia remains intact (Fig 112). If these criteria are present, hemostats are removed, residual bleeders, if any,

are caught and tied with very fine plain catgut and the wound is then thoroughly irrigated with sterile saline solution. Adhesive retracting strips are removed and the skin is again prepared and redraped. With a full-curved cervix needle sutures of very fine alloy steel wire (no 35) are placed at short intervals starting at the distal end of the wound. The suture incorporates the deep areolar tissue on both sides as well as a wide portion of the postsacral fascia (Fig 113). It is technically easier to place all sutures before tying. Extremely fine wire is used for two reasons: (1) it causes practically no tissue reaction and (2) its low tensile strength insures against excess tension in the wound. If the suture will not hold tension is considered excessive and primary closure in such a wound is thereby precluded. In deep defects a second superficial layer of no 35 wire sutures is used. All knots must be square tied three times, and cut on the knot to avoid sharp ends. Finally through and through vertical mattress sutures of no 28 wire are passed on large curved cutting needles starting 3 cm from the near skin edge. The near end is then threaded on a fine cutting needle and the skin edges are approximated. The tension sutures are tied firmly over gauze rolls (Fig 114). A dry dressing firmly held by wide adhesive straps placed across the buttocks completes the procedure.

The patient may be partially ambulatory after the third postoperative day but sitting is forbidden. The primary dressing is retained until the tenth postoperative day. Tension sutures are then removed and an alcohol dressing is applied and firmly fixed with wide adhesive straps. Dressings are discontinued after the fourteenth postoperative day. To avoid pressure and tension on the fresh wound the patient is cautioned to sit only on the lateral portion of the thigh and buttock with the opposite leg crossed over the knee during convalescence and for some time thereafter. As prophylaxis 600 000 units of procaine penicillin and 1 Gm of streptomycin are administered the day before operation and daily through the fifth postoperative day.

Recurrence is often attributed to the fact that some portion of the sinus tract remained in the wound due to incomplete removal

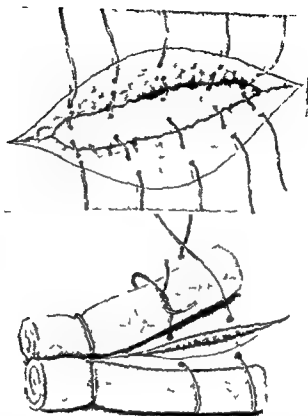


FIG 113 (*above*)—Closure after excision of cyst and sinus. Fine wire sutures incorporate lateral areolar tissue and postsacral fascia when tied deep portion of wound is approximated without tension dead space is eliminated adequate vascularity is assured and mobility of subcutaneous areolar pad is unimpaired.

FIG 114 (*below*)—Vertical mattress tension sutures of wire tied over gauze bolsters firmly approximate body of wound.

subsequently this acted as a nidus for reactivation of the infection. This factor is probably of no great importance because surgeons are generally cognizant of this pitfall in technique and are extremely careful to remove completely all sinus tracts.

In most of our cases at St. Albans Naval Hospital with recurrence after unsuccessful primary suture we were impressed with the uni-

form location and pathology of the lesion (Fig 115) Well healed scars were found over the sacrococcygeal region but typically, a draining sinus opening was present at the distal end of the scar, deep in the natal fold Through this opening a probe could be passed into the sinus which extended cephalad deeply under the scar directly over the sacrum At secondary operation when the healed cicatrix formed by the previous approximation of subcutaneous areolar tissue and skin was divided a deep sinus tract was exposed containing friable violaceous chronic granulation tissue Dwight (12) reported identical findings in recurrent sinuses following failure of the Shute type of gluteal muscle flap primary closures

Such uniform failure of permanent healing must be ascribed to a fundamental defect in surgical technic That these poor results were not due to haphazard technic by individual surgeons is shown by the fact that our 43 recurrent primary cases were sutured by many different surgeons in many parts of the country presumably with a variety of individual methods and various suture materials Furthermore in many cases recurrence took place many months after operation some after one or two years Interestingly enough only the deepest portion of the repair the region directly overriding the sacrum and coccyx broke down The skin and subcutaneous tissue which formerly contained the bulk of the pilonidal disease remained firmly healed, the defect left by excision having been replaced by a firm mobile well healed scar In a few exceptional cases a secondary sinus opening penetrated the proximal portion of the scar

The uniformity of the lesion found in our recurrent cases suggests that fundamental factors in wound repair had been disregarded Proof that infection was not the chief factor is supplied by the facts that the wounds in the subcutaneous tissues healed perfectly and that breakdown of the wound often occurred months after operation and apparent cure In a review of the literature regarding the treatment of pilonidal disease by primary suture it becomes obvious that many do not have a clear concept of the anatomy of the region Thus many describe the removal of the cyst and all possible sec

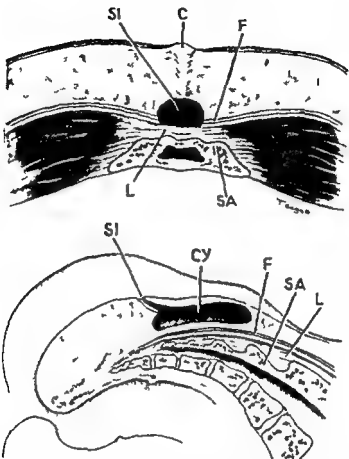


FIG 115 (*above*)—Typical recurrent sinus after unsuccessful primary closure SI sinus C cicatrix F postsacral fascia L posterior sacrococcygeal ligament SA sacrum

FIG 116 (*below*)—Anatomy of pilonidal sinocystic disease SI sinus CY cyst F postsacral fascia SA sacrum L posterior sacrococcygeal ligament

ondary sinuses down to the sacral fascia. The impression given is that the postsacral fascia and the posterior sacrococcygeal ligament are analogous. This is far from true.

Actually the postsacral fascia delimits the ventral extent of the very vascular loose fatty areolar subcutaneous tissue to which this

thin but firm fibrous fascial membrane is intimately attached. A potential space separates the postsacral fascia from the posterior sacrococcygeal ligament as demonstrated by the relative mobility of the fascia which slides freely over the ligament when the fascia is exposed at operation. The posterior sacrococcygeal ligament consists of a firm tendinous shiny relatively avascular ligamentous structure which is intimately attached to the periosteum of the sacrum and coccyx and laterally is confluent with the fascia of the gluteal muscles (Fig. 116). The distinction between fascia and ligament must be clearly understood as therein lies the key to successful surgical treatment.

In dissection of the cysts and sinuses down to the sacral fascia, it is probable that dissection is unknowingly carried through the postsacral fascia thereby exposing the posterior sacrococcygeal ligament. The accurate closure which follows with care to avoid dead space and tension on the suture line results in a sutured wound which presumably accurately approximates the tissue in layers arranged as follows: (1) posterior sacrococcygeal ligament to areolar tissue and fascia; (2) areolar tissue to areolar tissue; and (3) skin to skin. It is a well-established physiologic fact that primary healing of sutured wounds depends on a rich vascular bed in each of the approximated tissues. This criterion is satisfied in both the skin and the subcutaneous areolar tissues of the sacral region so that excellent healing is expected and was in fact uniformly found in our cases. However, it is physiologically unsound to expect firm union when richly vascular fatty areolar tissue is approximated to the posterior sacrococcygeal ligament, a tendinous fibrous and practically avascular structure. Similar principles apply to the repair of inguinal hernia as pointed out by Gallie and LeMesurier (13) who stated that side to side suture of muscle (vascular) as in the suture of the internal oblique muscle to Poupart's ligament (avascular) results in union of practically no strength. Relative healing time is another factor to be considered. Firm healing of subcutaneous tissues requires about 10 days. One cannot reasonably expect strong union between areolar tissue and ligament in this short time. With re-

sumption of normal activity by the patient rigid fixation of the suture line is disturbed resulting in potential disruption of union and possible formation of a dead space

Infection is still another factor tending to disrupt firm union deep in the wound Despite rigid aseptic operative technic in excising pilonidal lesions in our series cultures of material taken from the depths of the wound just before closure uniformly showed growth of septic organisms In a small series of cases we grew hemolytic *Staphylococcus albus* in 10 *Streptococcus anaerobius* in six, hemolytic *Staph aureus* in two, and *Str fecalis* in one The mechanism of recurrence following primary closure in many cases proceeds somewhat as follows loss of fixation in the weak suture line between vascular subcutaneous areolar and avascular ligament in time results in a dead space, this becomes increasingly infected because of the presence of low grade septic organisms and manifests itself eventually as a deep sinus tract Although sepsis is invariably present in these cases it is of low virulence as shown by the fact that the well vascularized skin and subcutaneous areolar tissues heal firmly

PARTIAL CLOSURE—Widespread chronic pilonidal cysts connecting distally with a sinus tract opening over the lower coccyx are often found Lateral extension of the cyst necessitates wide dissection which leads to a large banjo shaped defect The width of the wound in its proximal portion precludes primary closure However in its distal portion primary closure is often feasible and readily accomplished The wide upper portion is then partially closed by removing wedges of subcutaneous areolar tissue on each side thus allowing the skin to be drawn partially together to minimize the defect The skin margins are secured to the postsacral fascia by interrupted mattress sutures of alloy steel wire leaving only a small central area to heal by granulation Patients so treated required four to six weeks for complete epithelization

EXTERIORIZATION—In civilian practice the majority of patients with pilonidal disease seek surgical care following repeated episodes of acute or subacute inflammation On examination the large cyst

widespread sinuses and inflammatory induration of the adjacent skin and subcutaneous tissues pose a problem as to the best method of treatment. En bloc excision would entail the sacrifice of a large area of tissue and leave an extensive defect. Morbidity is prolonged and after final healing there is a wide tender scar over the sacrum and coccyx which is constantly subject to trauma.

In many respects this type of pilonidal disease is structurally similar to chronic perianal fistulas and abscesses. All three condi-

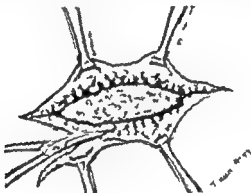


FIG 117—Extirpation for infected and extensive sinocystic disease

tions can be similarly treated by unroofing and providing wide open drainage. Buie pointed this out in 1937 when he advocated marsupialization instead of wide excision for pilonidal sinocystic disease.

In this obviously infected field saddle block spinal anesthesia is used. A grooved director is inserted into the main cyst and the overlying tissues are divided on the director to expose the cyst cavity. The sides are widely saucerized thereby unroofing the cyst. Associated sinus tracts are explored with a grooved director and the overlying tissue is divided to expose the tract throughout its length. The edges are saucerized to form a shallow groove. The granulation tissue in the base of the wound is gently removed with a spoon curet and bleeding is controlled by suture if necessary (Fig 117).

Buie and Curtiss (3) advised marsupialization of the wound by

suturing the skin to the base lining of the cyst with a running suture. However, I must agree with Brust and Sarner (15) that the cyst lining is often necrotic and so friable that marsupialization is not possible. In my experience there has been little difference in the healing time in patients treated by marsupialization and in those merely exteriorized. For this reason I have abandoned the marsupialization phase of Buie's exteriorization operation.

Great care must be taken to insure that pocketing does not occur at the inferior pole of the wound. Delayed healing in most cases is due to insufficient drainage as a result of such pocketing. The skin distal to the wound must be cut back far enough to allow very shallow saucerization (16).

Upon completion of the operation, the wound is packed with rinsed iodoform gauze and the superficial dressings are held firmly in place with adhesive straps. Patients are ambulatory on the first postoperative day, and sitz baths may be taken. The iodoform pack literally falls out on the fourth postoperative day to reveal young granulation tissue in the base of the wound. The anal douche is utilized several times daily until epithelization is complete. Healing averaged five weeks in my patients (Fig. 118).

Recurrence following excision in which the wound was left open to heal by granulation was less common. It was probably due in these cases, to insufficient wound drainage and to lack of careful postoperative wound toilet which resulted in the bridging of granulations and subsequent formation of infected sinuses.

ACUTE ABSCESSSES—Acute pilonidal abscesses are relatively common. They should be incised immediately if fluctuant thereby affording external exit for pus under tension and so limiting spread of the lesion. Wet dressings are applied for about three days to favor subsidence of wound induration. A secondary operation is then performed. After the wound is explored with probe and gloved finger it is saucerized by liberal excision of skin and subcutaneous tissues around the wound cavity. An exteriorized wound results which is treated as outlined earlier. Antibiotics are not used routinely because drainage is adequate.

TREATMENT OF RECURRENT PILONIDAL DISEASE—Recurrence is the bugbear of every surgeon who deals with sacrococcygeal pilonidal disease. Reports of successful primary closures were based in most instances on short postoperative observations usually in military personnel on whom late follow up results were unobtainable. That many lesions classified as cured later broke down is indicated



FIG 118 —Healed wound six weeks after exteriorization operation for chronic sinocystic disease with extensive and multiple lateral sinuses

by the fact that at the US Veterans Hospital the Bronx New York in 1946 an estimated 30 per cent of admissions for pilonidal disease were for recurrence after definitive surgical treatment during recent military service. For the same year the rate of admission to St Albans Naval Hospital for recurrence approached 40 per cent in veterans.

Most surgeons believe that primary closure fails as a method of treatment for recurrent pilonidal disease. The accepted treatment is some type of open technic which will provide adequate wound exteriorization. The importance of drainage in these wounds is dem-

onstrated repeatedly in extensive cases with defects from the tip of the coccyx to well up on the sacrum. The portion of the wound over the sacrum healed well and epithelized rapidly. However in the lower portion in which drainage was obstructed by apposition of the buttocks especially with the patient standing healing was sluggish and final epithelization was considerably protracted. In one patient who had had three operations for his recurring lesion the distance from the depth of the pilonidal wound in the coccygeal region to the surface of the buttocks measured 8 cm with the patient in the erect position. In this type of wound adequate drainage is mechanically impossible. Chronicity of the lesion in the recurrent cases produced excessive fibrous tissue in the granular bed of the wound resulting in a progressively inadequate blood supply to local tissues a factor which tends to retard healing (17).

Experience has revealed the difficulties in the surgical cure of patients with multiple recurrences. In several recalcitrant cases the local lesion was excised again so as to obtain a fresh clean wound. On this fresh wound a split thickness skin graft taken from the lumbar region of the back was fitted and sutured then held firmly in place with a sponge pressure dressing. In the few patients so treated the results were eminently successful. Skin grafting therefore would seem to be a solution of this very troublesome problem. Rank has described a similar procedure (18).

REFERENCES

- 1 McHardie M Pilonidal sinus *Ann Surg* 107 389 1938
- 2 Miller J M *et al* Streptokinase and streptodornase in the treatment of pilonidal cysts *U S Armed Forces M J* 2 1423 1951
- 3 Buie L A and Curtiss R K Pilonidal disease *S Clin. North America* 32 1217 1952
- 4 Gage M Pilonidal sinus. An explanation of its embryologic development *Arch Surg* 31 175 1935
- 5 Koostira H P Pilonidal sinuses *Am J Surg* 55 3 1912
- 6 Fox S L The origin of pilonidal sinus *Surg Gynec & Obst* 60 137 1935
- 7 Patey D H and Scarf R W Pilonidal sinus in a barber's hand *Lancet* 2 13 1918
- 8 Smith T E Anterior or perineal pilonidal cysts *JAMA* 136 973 1918
- 9 King E S J The nature of the pilonidal sinus *Australian & New Zealand J Surg* 16 182 1947

- 10 Hodges R M. Pilonidal sinus Boston M & S J 103 485 1880
- 11 Granet L. and Ferguson L. K. Pilonidal disease Management of cysts sinuses and abscesses in naval personnel Am J Surg 80 139 1945
- 12 Dwight R W. Pilonidal sinus A M A Arch Surg 64 438 1952
- 13 Gallie W L. and LeMesurier A B. The use of free transplants in the treatment of hernia Arch Surg 9 516 1921
- 11 Ferguson L. K. Pilonidal cysts treatment by excision and primary closure Ann Surg. 101 169 1935
- 15 Brust J C. M. and Sarner J B. Pilonidal cyst New York State J Med 48 2138 1948
- 16 Blaisdell P C. The healing open pilonidal wound J A M A 133 916 1947
- 17 Granet L. and Palmer B. The management of recurrent pilonidal disease Tr Am Proctol Soc 45 510 1946
- 18 Rank B K. Plastic principles in common surgical procedures Australian & New Zealand J Surg. 14 14 1944

CHAPTER EIGHTEEN

Miscellany

PARASITIC DISEASES OF RECTUM

PARASITIC DISEASES of direct concern to the proctologist are three amebiasis (*see* Chapter 11), schistosomiasis and enterobiasis

SCHISTOSOMIASIS

The schistosomes are blood flukes whose hosts are snails. The species which infest man are the oriental *Schistosoma japonicum*, and the *Schistosoma mansoni* which originates in Africa, South America and the West Indies. Man is infected by penetration of the skin by free swimming cercariae and subsequent growth of the immature worms in the blood vessels. The ova (Fig. 119) are deposited in the venules of the intestine and rectum; they penetrate the venous capillaries and the mucosa and are excreted from the body in the feces (1).

Pathologic lesions resulting from schistosome infestation include hepatitis, hepatic cirrhosis, splenomegaly, chronic fibrositis of the lungs and mesenteric lymphadenitis. In the rectum and intestine the invading ova produce an intense cellular infiltration of the mucosa and formation of papillae. The minute papillae rupture and are seen occasionally as small superficial ulcerations.

Characteristic symptoms are attacks of diarrhea with varying amounts of bloody mucus in the stools. A rectal granuloma and cryptitis due to *S. mansoni* has been reported (2). Systemic symptoms consisting of fever, anorexia, abdominal pain and loss of

weight are usually mild. Cases of overwhelming infection have been described in the Orient and in Africa.

The finding of characteristic ova in the feces is diagnostic of the disease. The ovum of *S. mansoni* has a mean size of $155 \times 66 \mu$, is oval and elongated with a large spine (20μ) emerging later



FIG. 119—Ova of *Schistosoma mansoni* in rectal mucosa. (From B. W. Warner, *Am. J. Surg.* 57: 168, 1912.)

ally. The ovum of *S. japonica* is $90 \times 65 \mu$, oval with a small curved spine emerging laterally. In a simple method of diagnosis a small superficial segment of mucosa is obtained from the edge of a valve of Houston by biopsy punch. After fixation for a few minutes in saline solution the tissue is placed on a slide and pressed flat with a cover slip. Typical ova are easily seen under the low power of a microscope.

Fuadin, the therapeutic drug of choice, is administered intra

muscularly. The first three doses 15, 35 and 5 cc., are administered on successive days, and thereafter 5 cc. is given every second day until a total dosage of 100 cc. has been given. Should relapse occur, a second course of treatment must be given after a rest period of three months.

ENTEROBIASIS

The small round pinworm, *Enterobius vermicularis* (*Oxyuris vermicularis*), inhabits the cecum and adjacent portions of the intestines of man. The female worm averages 10×0.3 mm. has a rigid body and a long curving tail. A gravid female contains an average of 11,000 ova. The male is rarely seen, probably because he dies soon after copulation. The mature female worms move irregularly in the intestine toward the rectum and are passed with the feces. The females migrate from the anus and crawl actively around the perianal region, sometimes even entering the vagina. The eggs are deposited when the worm is stimulated by contact with air. If the worm dries it explodes, showering thousands of ova on the anal verge.

Myriads of embryonate ova are deposited on the perianal skin of an infected individual during the night. The itching caused by the crawling worms leads to scratching and consequent contamination of the hands. The ova are transferred to the mouth directly from contaminated hands or secondarily through contamination of food and drink. An intermediate host is not necessary to complete the life cycle of pinworms.

Pinworms have been found in specimens of appendix removed in cases of subacute appendicitis. They have been found in an ischio-rectal abscess (3). In one of my cases that of a 62 year old man operated on for pyogenic cryptitis, the wound healed sluggishly so that a small portion of the wound remained free of epithelium four weeks after operation despite adequate drainage and meticulous wound toilet. On one occasion a single female pinworm was found with its head burrowed into the granulation tissue in the base of the wound. No other worms were seen on the skin or in the anus at this

time Treatment was purposely withheld On his next visit, five weeks postoperatively, a female pinworm was again present in the wound and ova were found on the perianal skin A course of gentian violet was administered following which ova could no longer be demonstrated and the wound epithelized promptly A contact source of infection could not be found in the immediate family nor in several of his frequently visited grandchildren In this case it is possible that pinworms burrowing into the granulation tissue specifically prevented epithelization of the wound

The prevalence of *Enterobius* infestation in children is well known with the concomitant infestation of adults in households of infected children The diagnosis is quickly made by inspecting the anal orifice of suspected children at some time during the night or early in the morning while the worms are still outside the rectum All stools of suspects should be visually inspected for the presence of the small curved ivory-colored pinworm

The Scotch tape swab simplifies the laboratory diagnosis A strip of Scotch tape with adhesive surface exposed is folded over the end of a wooden tongue depressor The perianal region is blotted with the Scotch tape After removal the tape is placed on a slide adhesive side down and examined under low power for ova

Brady (4) emphasized two important considerations in treatment First the infestation must be considered a family disease Unless all members of the family of a patient known to be infested are simultaneously treated the maturation and migration of a single female in an untreated individual may reinfect the entire household Second the treatment must be continued over a long enough time to insure death of all pinworm ova about the house in dust and on linen From his experience Brady believed that 23 days satisfactorily accomplishes this result

Gentian violet by mouth still appears to be the standard drug for treatment despite frequent manifestations of gastrointestinal irritation which include nausea vomiting abdominal pain and diarrhea Enteric coated tablets are available as $\frac{1}{2}$ and $\frac{3}{20}$ gr Enseals The dosage for adults is 2 Enseals of $\frac{1}{2}$ gr an hour before

not marked Diarrhea was present in 35 per cent, proctalgia in 32 per cent, and tenesmus in 24 per cent of the patients In four patients, a rectovaginal fistula developed

The sigmoidoscopic findings generally depend on the lapse of time since irradiation Early lesions are characterized by induration of the bowel wall, edema and injection of the mucosa Telangiectatic areas are seen and the mucosa is friable Necrosis of the mucosa persists as a chronic ulcer which is oval or irregularly circular usually single, and involves one or more layers of the rectal wall The ulcer has a necrotic base covered by grayish membrane which bleeds easily and is ordinarily located on the anterior wall of the ampullary rectum Stricture results from fibrotic infiltration throughout the wall of the rectum and is seen as a smooth and symmetrical narrowing of the bowel It may involve the rectum or the sigmoid In Buie's cases the aperture averaged 1.3 cm. in diameter

TREATMENT—The entirely symptomatic treatment is along broad, general lines based on the findings in individual cases Ascorbic acid is administered in daily doses of 500 mg Self administered cleansing enemas of small amounts of warm water help to relieve tenesmus by removing irritating mucus and fecal matter Rectal instillations of 2 oz. of crude cod liver oil are advisable A bland diet, sedation as required and frequent periods of bed rest are essential Local surgical treatment for complications such as rectovaginal fistulas must be deferred until enough time has elapsed to be certain that complete clinical cure of the primary genital cancer has been obtained Tissue damaged by irradiation may show no propensity to heal A colostomy for severe stricture ulceration or fistula may sometimes be required

TRAUMA

The rectum is well protected by the bony pelvis its associated fascia and muscle structures so that injuries from external violence are relatively infrequent Perforations caused by sigmoidoscopy are discussed in Chapter 2 The literature contains a few reports of penetrating injuries caused by bizarre accidents most of which are

due to impalement by sharp objects such as sticks tree branches corn stalks and iron fence pickets Gunshot or shrapnel wounds occur frequently during war

Siler and Bebb (12) reviewed the subject and classified wounds of violence affecting the lower bowel and perineum into five anatomic categories

- 1 The urogenital region the urethra and bladder suffer injury
- 2 The anal region suffers lacerations involving the anal musculature and the perianal regions
- 3 The ischiorectal fossa may be directly involved in wounds or may be contaminated through wounds extending laterally from injuries to the rectum
- 4 The supralelevator spaces are similarly affected Infection may extend to involve the retroperitoneal space with overwhelming sepsis and death

5 The rectum and sigmoid can be perforated by impalement or by gunshot wounds Industrial workers have suffered rupture of the lower bowel from sudden blasts of compressed air directed at the anus either accidentally or more often by practical jokers (13 14)

Perforations of the rectum by enema tips and of the colon by faulty administration of enemas have been frequently reported In these cases shock circulatory collapse and peritonitis rapidly ensue

TREATMENT—Patients with wounds of the perineum anus and buttocks should be hospitalized immediately after first aid measures have been administered Diagnostic procedures are then instituted to determine shock blood loss and bladder and peritoneal involvement Catheterization and urinalysis will usually show whether the bladder or urethra is injured Flat roentgenograms in the lateral recumbent position will indicate the presence or absence of pneumoperitoneum If the latter condition is present bowel perforation is assumed Sigmoidoscopy reveals traumatic lesions in the rectum and distal sigmoid Should there be the slightest suspicion of bowel perforation in impalement injuries or in gunshot wounds exploratory laparotomy must be performed without undue delay

In all cases shock and blood loss must be treated Tetanus anti

toxin must be administered routinely. Penicillin, streptomycin and intravenous sulfadiazine are given in large doses as an initial measure. If the bladder is involved, an indwelling catheter is inserted as a temporary measure. Wounds of the perineum are cleansed, foreign bodies are removed, and wound edges are widely saucerized for drainage. The best procedure for treating lacerations of the sphincter muscles must be individually evaluated. Clean, simple tears may be approximated immediately following local wound toilet. Ragged and necrotic lesions are best treated by debridement and delayed repair. Wounds of the ischiorectal space are explored for foreign bodies and the wound tract is exposed widely to provide adequate drainage. In deep perineal wounds, constant irrigation with Varidase (streptokinase streptodornase) may be very helpful in promoting clean healing (15).

Perforating wounds of the posterior ampullary rectum can be exposed through an incision made parallel to the coccyx and sacrum, supplemented by coccygectomy if necessary. Following suture of the rectum, a temporary colostomy should be established and the external fossa adequately drained.

Clinical signs of peritonitis and x ray evidence of pneumoperitoneum indicate high bowel perforation early and complete abdominal exploration is necessary in these instances. The reparative procedure depends on the findings in each case. Pilcher (16) reported his experience with over 200 cases of perforating war wounds of the colon and rectum. In almost all cases the perforated bowel was exteriorized by a Mikulicz type of procedure which was deemed far safer than intra abdominal closure under field conditions. In gunshot wounds involving the rectum, a thorough abdominal exploration is made to uncover the possibility of other visceral perforations. The rectal wound is then treated by primary or delayed closure as determined by the local condition.

Support with blood, electrolytes, antibiotics and chemotherapy must be maintained postoperatively and the patient must be continuously observed for the onset of such complications as sepsis, intestinal obstruction and electrolyte imbalance. In traumatic lesions

involving the lower bowel, bold measures based on sound surgical principles and instituted early should prevent disastrous consequences

EXTRARECTAL TUMORS

During a digital examination of the rectum an abnormal extra rectal mass is occasionally palpated. The mass may be inflammatory or it may originate in neoplastic disease of adjacent viscera.

Jackman and Anderson (17) have reported 27 cases of prostatic carcinoma in which the rectum was involved. Findings in these cases included (1) external pressure with obstruction of the rectal lumen (2) an hourglass constriction of the rectum and (3) infiltration of the rectal wall.

In another report Jackman, Clark, and Smith (18) collected 114 cases in which tumors of the retrorectal space were found. Inflammatory tumors accounted for most of the retrorectal masses. These lesions consisted of chronic abscesses of the retrorectal space many with external fistulas and chemical granulomas which were sequelae of sclerosing therapy for hemorrhoids or prolapse. Congenital tumors included teratomas, dermoid cysts, chordomas, and a meningocele. Neurogenic tumors were classified as neurofibromas, ependymomas, and a neurilemmoma. There were five tumors of osseous origin exclusive of chordoma invading the retrorectal space.

Chordomas occurred in 14 cases, almost half of all congenital tumors encountered. These were classified as congenital tumors because of their origin from fetal remnants of the notochord. Of the spinal column chordomas, 70 per cent occur in the presacral region. In a review of the literature, Littman (19) found reports of 168 chordomas. Chordomas produce severe pain and other symptoms caused by the growth of the characteristic lobulated gelatinous masses which progressively destroy the adjacent bony sacrum. In the roentgenograms this appears as an erosive destructive process and is diagnostic. Needle biopsy reveals a characteristic vacuolation of the cells with production of excessive mucus.

Treatment is necessarily surgical. Because of the multiple manifestations produced by this bizarre tumor, MacCarty *et al* (20)

have shown that the lesion can be best treated by a cooperating team consisting of a neurosurgeon, an orthopedic surgeon and a general surgeon. These investigators have treated several cases by radical removal of the tumor and, in addition, removal of the sacrum and coccyx below the second sacral vertebra. The sacrifice of the third and fourth sacral nerves was unavoidable but no significant vesicular or rectal dysfunction resulted. They reported excellent short term results following their radical excisional procedure.

FECAL IMPACTION

An intraluminal rectal or lower sigmoidal mass consisting largely or entirely of inspissated feces is known as fecal impaction. The mass is so large and so firm that for one reason or another it cannot be voluntarily expelled through the anus. Individuals subject to rectal constipation occasionally have a fecal impaction as a result of desiccation in the too long retained fecal mass and its constant augmentation in the voluminous posterior rectal pouch. Dietary materials such as cellulose from fruits and vegetables, nuts, seeds, lime silt from dairy products, and insoluble drugs such as salol, bismuth and barium may form the core on which the impacting bolus is constructed.

Fecal impaction occurs frequently in feeble senile individuals and in bedridden invalids. It is often encountered after gastrointestinal or other abdominal surgery. It must be vigorously guarded against following ophthalmic operations for example removal of cataracts and operations for retinal detachment, in the postoperative period absolute immobility is mandatory and even the expulsive effort of normal defecation is banned. Impaction is common in patients with long standing anal ulcers and associated anal stenosis. Occasionally impaction occurs in the early period following surgery for benign anorectal lesions.

Symptoms vary widely. Obstipation is usually the first symptom. Subsequent fixation produces bowel movements in the form of frequent mushy or liquid dejections. These are accompanied by tenesmus and a sense of incomplete evacuation which in reality it

■ The liquid bowel content resulting from the laxative passes around the obstructing impacted bolus and ■ expelled as ■ diarrhetic stool When frequent yet unsatisfactory bowel dejections occur after anorectal surgical procedures a digital examination must be performed to determine the possible presence of an impaction

Chronic impactions are characterized by ■ sense of discomfort pressure or actual pain in the rectum pelvis or perineum Finally ■ result of mucosal erosion by the inspissated intrarectal mass tenesmus with bloody mucoid dejections ensues A simple digital examination performed preferably with the patient in Sims's position establishes the diagnosis

Treatment must vary to suit individual circumstances In early postoperative impactions the mass is usually large and soft In such cases a mild soap and water enema administered after careful insertion of a blunt-end rectal tube is a simple and effective method of affording relief The buttocks must be held closed around the rectal tube by the gloved hand so that all of the enema fluid can be instilled The vigorous defecation reflex stimulated by the enema results in a forceful expulsive effort which overcomes voluntary sphincter spasm and delivers the fecal mass along with the enema fluid If the mass is not expelled but remains impacted it becomes necessary to break up the fecal mass with a lubricated and gloved finger following which the enema is repeated A similar technic ■ effective in dealing with soft impactions in the senile in celiotomized patients postoperatively and in many patients who have acute impactions due to the barium sulfate administered for upper gastrointestinal examinations Patients having gastrointestinal series should routinely be advised to take a daily enema for three days following barium examination as a prophylactic measure against impaction

In the occasional patient in whom the impaction is extremely firm hard or of calcific consistency digital disruption ■ ineffectual The mass can often be delivered with the aid of the encircling index finger following anal dilatation under regional anesthesia This maneuver is best performed with the patient in lithotomy position and with an assistant exerting downward manual pressure from

the suprapubic region, so fixing the rectum and sigmoid in the pelvis. Should this maneuver fail a large lumened tubular operating proctoscope is inserted, if necessary after posterior sphincterotomy. The calcified bolus is grasped with a cervical tenaculum and fragmented with a lithotrite or a similar crushing instrument. The fragments are then removed piecemeal.

Oil retention enemas are usually ineffectual in the treatment of established impactions. Strong peroxide enemas have been given for fecal impaction, but their caustic action on the mucosa of the lower bowel makes them dangerous. Severe proctitis with sloughing of the mucosa has resulted from peroxide enemas (21).

FOREIGN BODIES

The natural conformation of the rectum admirably suits it for its function as a repository for the fecal bolus. The contour of the rectum likewise renders it particularly favorable for the arrest of foreign bodies which were swallowed or the retention of objects accidentally or intentionally inserted through the anus. Occasionally a foreign body develops in some portion of the gastrointestinal tract and passes down to lodge in the rectum. Gallstones, fecaliths formed in and subsequently extruded from diverticular pockets and coproliths are the commonest examples. These products not infrequently form the core of a calcified impaction. Small foreign bodies such as fruit pits or stones, chicken or fish bones, pins, toothpicks, coins and buttons may be accidentally swallowed especially by children. They may lodge in the anal crypts or above a valve of Houston leading to local mucosal erosion or even a perianal or perirectal abscess. These objects are easily removed by digital manipulation or grasped and removed following visualization through an endoscope.

Psychiatric patients swallow needles, nails, screws, hairpins, glass fragments and sundry other objects in smaller or larger quantities over an extended period of time. Oddly enough some of these objects make their way unobstructed through the gastrointestinal tract to lodge in the sigmoid or rectum. They may erode or per-

forate the wall of the sigmoid or rectum with ensuing peritonitis or pelvic abscess

The literature is well documented with reports of foreign bodies of the most varied and marvelous character which were found in the lumen of the rectum or lower colon. These include bottles of various shapes and sizes, electric light bulbs, drinking glasses, tools, cutlery, door knobs, various fruits, and phallus shaped objects of various kinds, such as corn cobs, candles, cow horns, and wooden sticks. Such objects may slip into the rectum while being used as a mechanical aid in controlling rectal prolapse or prolapsed bleeding hemorrhoids. They are sometimes lost into the rectum of an anal erotic individual who was using the object for anal masturbation, often during depraved orgies induced by alcohol or drugs. Sadists have forced various foreign bodies into the rectum of their victims (22). Tuttle (23) reported many bizarre instances of rectal foreign bodies, the case histories of which make interesting reading.

More frequently, cases are encountered in which foreign bodies have unintentionally slipped into the rectum during nursing procedures. Rectal thermometers and hard rubber enema tips are frequently lost, especially in children. These objects are easily removed through an endoscope.

Symptoms resulting from foreign bodies in the lower bowel vary with the individual circumstances. They may be lacking when the object is small and smooth, the patient reporting presence of the object in the rectum from his direct knowledge of the manner of its insertion or ingestion. Sharp objects traumatize the rectum or anus, causing pain or bleeding, or both. Perforation of the rectum may occur, manifested in the clinical signs of perirectal abscess, or of pelvic or generalized peritonitis. Digital and endoscopic examination establishes the diagnosis of an intraluminal foreign body and allows an evaluation of the best way to remove it.

Large foreign bodies often are difficult to remove. Many ingenious methods have been used, and a number of patients have died of peritonitis which followed difficult and traumatizing extractions. In general, all planned extensive manipulations should be

preceded by preparation of the patient with suitable antibiotics. The sphincter must be completely relaxed by a regional anesthetic supplemented by posterior sphincterotomy, if necessary, to obtain space for manual manipulation. If it is found that a large foreign body, such as a glass is wedged in the ampulla or the rectosigmoid, operative approach should be by posterior proctotomy following coccygectomy. Some of the methods are fully described by Bacon (24).

REFERENCES

- 1 Belding D L. *Textbook of Clinical Parasitology* (New York: Appleton Century Company 1942)
- 2 Warner B W. Anorectal manifestations of *S. mansoni* infestation. *Am J Surg* 57: 168 1942
- 3 Marshall R G and Wood Q L. Ischioanal abscess caused by *Oxyuris vermicularis*. *Northwest Med* 37: 180 1938
- 4 Brady F J. Pinworm infection. *Am Pract* 1: 583 1947
- 5 Bumbalo T S and Gustins F J. Treatment of pinworm infection in children. *Postgrad Med* 14: 83 1953
- 6 Speare G S. Melanosis coli. *Am J Surg* 82: 631 1951
- 7 Bockus H L, *et al*. Melanosis coli. *JAMA* 101: 1 1933
- 8 Buie L A and Malmgren G E. Factual proctitis: A justifiable lesion observed in patients following irradiation. *Internat. Clin.* 3: 68 1930
- 9 Craig M S Jr and Buie L A. Factual (irradiation) proctitis. *Surgery* 25: 472 1949
- 10 Aune E T and White E V. Gastrointestinal complications of irradiation for carcinoma of uterine cervix. *JAMA* 147: 831 1951
- 11 Wiley H M and Sugarbaker E D. Roentgenotherapeutic changes in the small intestine. *Cancer* 3: 629 1950
- 12 Siler U E and Bebb K. Trauma to the perineum, anus, rectum and colon. *Am J Surg* 80: 652 1950
- 13 Block F H and Weissman M I. Pneumatic rupture of sigmoid. *JAMA* 86: 1397 1926
- 14 Burr C A V. Pneumatic rupture of the intestinal canal. *Arch Surg* 22: 872 1931
- 15 Beahrs O H and Jordan G I. Use of streptokinase and streptodornase in primary closure of the posterior wounds. *Proc. Staff Meet. Mayo Clin.* 27: 241 1952
- 16 Filcher L S. Wounds of the colon and rectum. *Mil Surgeon* 104: 188 1919
- 17 Jackman R J and Anderson J R. Proctologic manifestations of carcinoma of the prostate. *Am J Surg* 83: 491 1952
- 18 Jackman R J, Clark P L and Smith N D. Retrorectal tumors. *JAMA* 145: 956 1951
- 19 Littman L. Sacro-coccygeal chordomas. *Ann Surg* 137: 80 1953

- 20 MacCarty C. S., *et al* Surgical treatment of presacral tumors. A combined problem Proc Staff Meet. Mayo Clin. 27 73 1952
- 21 Pumphrey R. E. Hydrogen peroxide proctitis Am J Surg. 81 60 1951
- 22 Vogel F. Foreign bodies in the pararectum due to masochism Am J Psychotherapy 5 236 1951
- 23 Tuttle J F. *Diseases of the Anus Rectum and Pelvic Colon* (New York D Appleton & Company 1903)
- 24 Bacon, H. E. *Anus Rectum Sigmoid Colon* (3d ed. Philadelphia J B Lippincott Company 1949)

Index

A

- Abscess perianal**
apocrine 254
causes 84 108
in children incidence 66 f 72
pilonidal (acute) 302 303
treatment 314
recurring results of 87
symptoms and diagnosis 89
treatment 90 ff
incision for drainage 90
stem to-stern operation for 91
ff
tuberculous 239
treatment 241
types of 84
- Abscess supralevator**
diagnosis 93 f
surgical treatment 94
- Acid Mantle Creme** for pruritus ani 265
- Actinomycosis anorectal** 254 f
- Adenocarcinoma** 185 ff
see also Cancer of colon and rectum
classification 186 ff
method of spread 189 ff
pathology 185 f
- Adenoma colorectal**
and cancer 155 156 f
influence on treatment 160 f
incidence 153
papillary *see* Papilloma, villous
pathology 152
- Adenomatosis polypoid** 163 ff
- Alcohol**
infusion for pain of terminal cancer 205
injection for pruritus ani complications 268
nerve block (sacral) for pain of terminal cancer 205
- Alimentation parenteral** for intractable ulcerative colitis 221
- Amebiasis** 230 ff
diagnosis 231
treatment 231 f
amebicides and antibiotics in 232
- Anatomy**
of anal canal 13 ff
relation to anal fissure 103 f
of anal crypts and ducts 83
anorectal
blood supply 20 ff
comparative in child and adult 67
lymphatic system 22 ff
nervous system 24 f
of apocrine glands 253
of rectum 15 ff
- Anesthesia** 39 ff
for abscess incision 90
caudal epidural block 46 ff
for endoscopy in children 68
general 53

Anesthesia (cont)

- infiltration 40 f
 - for acute hemorrhoidal disease 120
 - for coccygodynia 274 f
 - contraindicated in extensive surgery 41
- local of prolonged duration, 44 ff
 - for anal fissure 105 f
 - nonoleaginous drugs for 46
 - oil soluble drugs for 44 f
- local reactions to—prevention and control 42 f
- local of short duration 39 ff
 - infiltration 40 f
 - topical 39 f
- spinal saddle block 48 ff
 - with hyperbaric solutions 49
 - with hypobaric solutions 49 f
- topical ointments for 57

Anoderm 14**Anomalies**

- anorectal classification 72 ff
- extrarectal associated 75

Anoplasty for chronic fissure 109 ff**Anorectoplasty**

- for complicated hemorrhoids 136 ff
- postoperative care 142 ff
- postoperative complications 144 ff

Anorectum

- anatomic relationships 13 ff 67
- dyscrasias of 272 ff
 - neurogenic (bizette) 277 f
- examination 26 ff
 - bidigital in hemorrhoidectomy 129
 - in children 68
 - digital method 29 f
 - endoscopic instruments for 32 ff
 - inspection 29
 - symptoms indicating 26 f
 - topical anesthesia for 39
- exposure for hemorrhoidectomy 128 f
- infectious diseases of 238 ff
- lymphatic system of 22 ff

- malformations of 72 ff
- musculature 16 ff
- nervous system of 24 f
- pyogenic infections of 82 ff
- vascular system of 20 ff

Anoscope use of 32 f**Antibiotic therapy**

- for actinomycosis 255
- for amebiasis 232
- for bacillary dysentery 233
- diarrhea from 233 ff
 - pathogenesis 234
 - treatment 235
- for enterobiasis 322
- for hemorrhoidal disease (acute) 121
- for lymphogranuloma venereum 245
- for tuberculosis 242
- for ulcerative colitis 220

Antihistamines in pruritus ani therapy 266**Antipruritics in ointment form 57****Anucaine**

- for anal fissure analgesia 106
- for long lasting analgesia 44

Anus *see also* Anorectum specific conditions

- anatomy of 13 ff
- comparative in child and adult 67

- crypts and ducts spread of infection in 83 f

examination

- indications for 26 f
- inspection method 29
- topical anesthesia for 39

imperforate 73

- diagnosis 75
- fistulas in 72 74

injuries in 325**lesions of improper use of suppositories for 36****lymphatics of 22 f****musculature 16 f****nerve supply of 2 f****papillae hypertrophied 150 f**

Index

vascular supply 20 ff

Apocrine glands
anatomy 253

infection of 253 f

Aralen in amebiasis 232

Arteries of anorectal region 22

Ascorbic acid
for irradiation proctosigmoiditis 324
postoperatively for wound healing 59

B

Barbiturate for anesthetic drug reaction 42 43

Barium examination

of colon uses of 35

Gastrointestinal prevention of fecal impaction after 329

Biopsy

in colorectal cancer 193 ff

of colorectal polyps 158

importance 156

in tuberculous perianal infection 240

Bleeding

in cancer of lower bowel 176

from diverticula 293

after hemorroidectomy

at anal verge 145

massive from hemorrhoidal stump 145 ff

with hemorrhoids 117 f

rectal

in cancer diagnosis 176

in children 68 f

indication for examination 27

Blood transfusion

for posthemorrhoidectomy hemorrhage 145

in ulcerative colitis 220

Bowel

function in diverticulosis 293

movements

change in pattern and cancer 176

individual variations 26

in ulcerative colitis 215 219

obstruction from diverticulitis 295 f

perforation from diverticula 295 f

perforation—traumatic 325

from sigmoidoscopy 35 38

treatment 326

preparation of for instrumental examination 28

wall spread of cancer through 189 f

C

Cancer *see also* specific types
and adenomas of colon and rectum 155 ff

influence on treatment 160 f

of adjacent viscera, involving rectum 327

anal 180 ff

in chronic fissure 109

in long standing fistulas 101

metastatic spread of 182

pain of 177

results of treatment 182 183

types of 180

of colon and rectum

see incidence of 177

arising in polyp 161 f

classification 186 ff

colostomy for psychologic problems 172 f

delayed diagnosis factors in 174 f

diagnosis 177 ff

diagnosis by biopsy 193 ff

diagnosis by smear technic 195

incidence 173 175

method of spread 20 22 189 ff

multiple primary 195

operability 196 202

palliative Hartmann operation for 201 f

recurrent 203 f

surgical treatment 195 ff

symptoms 176 f

terminal management 204 f

treatment results 202 f

diverticulosis and 296

lymphogranuloma venereum and 246

Fistula (cont.)

- diagnosis 94 ff
- from diverticula 293 f
- horseshoe 86
- in imperforate anus 72 74
- multiple 87
 - staged surgical procedures for 100 f
- pathogenesis of 85 f
- primary opening—location
 - diagnostic methods 95 f
 - and surgical procedure 98 ff
- treatment 97 ff
 - en bloc dissection 98 ff
 - incision and drainage 97
- tuberculous 238 240
 - surgical treatment 241 f
- types of 85 f
 - anatomic relations 87

Fistulotomy 98 ff

- stem to-stem
 - technic 91 ff
- for tuberculous perianal abscesses 241

Foreign bodies in rectum 33b ff**Fres test in diagnosis of lymphogranuloma venereum 244****Fuadin for schistosomiasis 319****G****Galvanism**

- in anal stricture management 64 f

Gastrointestinal tract

- barium examination prevention of fecal impaction after 329
- reflex disturbances of cryptitis and papillitis in 88

Gentian violet for enterobiasis 321 f**Gonorrhea rectal 246 f****Goodsall's rule in outlining fistula tract 95****Granuloma**

- of colon (benign) 168
- inguinale involving anus 219 f

Gumma rectal 249**H****Hartmann operation for rectal cancer 201 f****Heat moist in postoperative wound care 63 f****Hematomas perianal 118****Hemorrhage see Bleeding****Hemorrhoidectomy**

- complications of 133 ff
- management 144 ff
- ligature operation 127 ff
 - Goodsall's triple ligature method 132

Hemorrhoids 114 ff

- classification of 116
- cold wet dressings for 64
- complicated 133
 - anorectoplasty for technic 136 ff
- diagnosis of 119
- etiology 114 f
- external location of 114
- external thrombotic (acute) 118
 - bistoury incision for 120
 - conservative treatment 120 ff
 - surgical excision 119 f
- internal 114
 - chronic, sclerotherapy for 122 ff
- internal thrombotic (acute) 118 f
 - management 121
- pathology 115
- "recurrent after hemorrhoidectomy 134

- simple surgical treatment 127 ff
- stump of
 - massive hemorrhage from 145 ff
 - pseudopolyp of 136

Hidradenitis suppurativa 253 f**Hirschsprung's disease**

- pathogenesis 76 f
- treatment 78 f

Hormone therapy

- for pruritus ani 266
- for ulcerative colitis 222

Hydrophilic colloids

- with kaolin in ulcerative colitis 219
- for laxation, 61
- in children 71

- in sigmoidal intussusception therapy 288
- Hydrotherapy 62 ff
- Hygiene anal for pruritus ani 263 ff
- Hyperpyrexia: in ulcerative colitis therapy 221
- Hysteria anorectal symptoms of 278

I

- Ileocolitis 214
- Ileostomy
 - bag use of 228
 - and colectomy for ulcerative colitis 223 ff
 - time of colectomy 226
 - reanastomosis after 228 f
 - rehabilitation of patient with 226 ff
- Impaction
 - calcified from foreign body 330
 - fecal 328 ff
 - symptoms 328 f
 - treatment 329 f
- Incontinence anal causes and prevention 289

Infections

- pyogenic of anorectum 82 ff
- specific of anorectum 238 ff
- spread of and lymphatic system 22

Instruments

- endoscopic 32 ff
- ring retractor in hemorrhoidectomy 128 f
- for sclerotherapy of internal hemorrhoids 125

Intestine *see* Bowel specific parts

Intussusception, sigmoidorectal

- diagnosis 286
- symptoms 285 f
- treatment 287 f

Itching *see* Pruritus

L

- Lactobacillus acidophilus in dietary therapy 59 226
- Lavage rectal 63
- in pruritus ani therapy 264

Laxatives

- anthracene causing melanosis coli 322
- in general therapy 59 ff
- hydrophilic colloid 61
 - for children 71
 - with kaolin in ulcerative colitis 219
- in sigmoidal intussusception 288
- mineral oil deleterious effects 60 82
- prune juice at 61
- saline 61
 - habitual use associated with cryptitis 82
- Levator ani 18 ff
 - massage for coccygodynia 274
 - spasm of 20
 - in coccygodynia 273
 - in proctalgia fugax 216

Lymphatics

- of anorectal region 22 ff
- spread of cancer by 189 191 f
- node resection, in cancer of colon and rectum 196 f 201

Lymphogranuloma venereum 242 ff

- diagnosis 244
- pathogenesis and clinical features 243 f
- treatment 245 f

Lymphoma anorectal benign 166 ff

Lymphosarcoma of colon and rectum 208

M

- Medicaments proctologic use 56 ff
- Megacolon 75 ff
 - idiopathic differentiated from Hirschsprung's 77 79
 - pathogenesis 75 ff
 - treatment, 78 f
- Melanoeplithelioma of anus 183 f
- Melanosis coli 322 f
- Melena in diverticulosis 293
- Methylcellulose laxatives 61
- Miltibis for amebiasis 232
- Mineral oil
 - habitual use and cryptitis 82

- Mineral oil (*cont*)
 as laxative deleterious effects 60
- Muscles anorectal 16 ff
 external sphincter 18
 levator ani 18 ff
 massage of 274
 spasm
 in anal fissure 103 f
 in coccygodynia 273
 diathermy in treatment 65 274
 in proctalgia fugax 275 f
- Mycoses perianal, 262
 with pruritus ani treatment, 265
- N
- Nerve
 block *see also* Anesthesia
 alcohol for pain of terminal cancer 203
 distribution and pain of anorectal lesions 25
 supply of rectum and anus 24 f
- Nervous system
 autonomic and ulcerative colitis 217
 sympathetic in idiopathic megacolon, 79
- Neurodermatitis perianal 263
- Neurotomy perianal subcutaneous
 technic 267 f
- Nitrites for spasm of proctalgia fugax
 277
- Nupercainal ointment for analgesia
 57
- Nupercaine for addle block 49 50 f
- O
- Ointments as vehicles for medica-
 ments 57 f
- Opium suppository 57
- P
- Pain
 of anal fissure 103 105
 anorectal
 conditions causing 27 f
 hydrotherapy for 62 f
 and nerve distribution 25
 mechanism
 in coccygodynia 272
 in proctalgia fugax 277
 postoperative control 44 f
 rectal idiopathic 275 ff
 in terminal cancer control 203
- Papillofibromas of anus treatment
 151
- Papillomas
 of anus 149 f
 villous of rectum and colon 162 f
 pathology 152
 symptoms 155
- Paraffinoma of rectum 166
- Parasitic diseases of rectum 318 ff
- Pentothal sodium for general anes-
 thesia 53
- Peritoneum relation to rectum 15 f
- Phenol-almond oil for sclerotherapy
 124
- Pile sentinel in chronic anal ulcer
 tract 107
- Pilonidal disease 298 ff
 etiology 299 ff
 hair in lesions of 301
 incidence 298
 interdigital 300
 pathology and clinical features 301
 ff
 recurrent
 cause 309 f
 treatment 315 f
- treatment
 dissection and partial closure 312
 dissection and primary closure
 304 ff
 externalization of wound 312 ff
 marsupialization of wound 313 f
- Pinworm infestation *see* Enterobius
- Podophyllin for anal warts dis-
 tances 252
- Polypoid multiple
 acquired 163
 hereditary 163 f
 treatment 165 f
- Polyps 152 ff
 biopsy of 158

- and cancer 155 156 f
 - in children 66 f
 - classification 152
 - pathology 152 f
 - symptoms 155
 - treatment
 - in children 69
 - electrosurgery of 64 157 f
 - local excision of 159 f
 - segmental resection for 159 f
 - type and location determining 158 f
 - with ulcerative colitis 217 f
- Pontocaine**
- ointment, for analgesia 57
 - for spinal saddle block 49
 - for topical anesthesia 40
- Pregnancy** in ulcerative colitis patients 219
- Procaine**
- infusion for pain of terminal cancer 205
 - for local anesthesia 40 43
- Procidentia, rectal** 281 f
- conservative therapy 282
 - surgical repair of 283
- Proctalgia**, 272 f
- see also* specific syndromes
 - fugax 275 f
- Proctitis**
- gonorrheal, 246 f
 - hypertrophic (circumscribed)
 - clinical and pathologic features 229
 - treatment, 230
 - of lymphogranuloma venereum
 - treatment 245
- Proctosigmoiditis**
- in children, from procidentia 286
 - irradiation (factitial)
 - symptoms 323
 - treatment, 324
 - localized 214
- Prolapse rectal**, 280 f
- in children, 66 f
 - external, therapy 71
 - factors favoring 69 f
 - internal 70 f
 - classification 280
 - mucosal 280 f
 - after hemorrhoidectomy 135
 - with hemorrhoids 117
 - in pruritus ani 262
- Prune juice** as laxative 61
- Pruritus**
- perianal from antibiotic therapy 235
 - physiology of 258
- Pruritus ani** 25 f
- alkalinity of rectal secretion in 260
 - dietary control 59 266
 - differential diagnosis 262 f
 - etiologic theories 257 258 f
 - neurogenic 268 f
 - psychotherapy for 270
 - pathology 258 f
 - recurrences of 257
 - treatment 263 f
 - anal hygiene in 63 263 f
 - complications of 268
 - systemic and local adjuncts to 46 266 f
 - wet dressings in 64
- Pseudopapillomas** of anus 149
- Pseudopolyps**
- at hemorrhoidectomy stump 136
 - treatment, 136
 - with ulcerative colitis 218
- Psoriasis perianal** 263
- Psychoneuroses** anal symptoms of 278
- Psychotherapy** for neurogenic pruritus ani 270
- Pteroyl triglutamate** for terminal cancer patients 205
- Q**
- Quinuride** for sclerotherapy 125 f
- R**
- Radium therapy** causing proctosigmoiditis 323 f
- Rectalgan** for topical analgesia, 57
- Rectocaine**
- for anal fissure analgesia, 106
 - for long lasting analgesia, 44

- Rectosigmoidectomy for rectal procidentia 283
 Rectum
 ampullary 15
 examination method 31
 anatomy of 15 ff
 bleeding from
 in children causes 68 f
 indication for examination 27
 cancer of 172 ff
 adenocarcinoma 185 ff
 location of and treatment method 197 ff
 comparison in child and adult 67
 examination
 digital method 29 f
 endoscopy 34 ff
 indications for 26 f
 foreign bodies in 330 ff
 injuries to 324 ff
 lavage 63
 lymphatic supply 22 f
 lymphoid hyperplasia (follicular) of 169
 malformations of 72 ff
 mucosa of
 pH of 260
 prolapse 280 f
 musculature 17 ff
 nerve supply 24 f
 parasitic diseases of 318 ff
 perforation of 324 ff
 peritoneal reflections of 15 f
 polypoid disease of 152 ff
 procidentia of 281 ff
 sigmoid prolapse into 284 ff
 treatment 297 f
 sphincteric anatomy of 15
 tumors of
 benign 150 152 ff
 carcinoid 210
 epidermoid 184
 tumors of adjacent viscera involving 327 f
 vascular supply 20 ff
 Roentgen therapy for pruritus ani 267
 complications of 268
 for squamous carcinoma of anus 182
 Roentgenography
 barium contrast uses 35
 in diagnosis
 of anal fistula 96
 of cancer 178 ff
 of diverticula of colon 293
 of imperforate anus, 75
 of ulcerative colitis 215
 S
 Schistosomiasis 318 ff
 Sclerotherapy
 for hemorrhoidal disease 122 ff
 indications and effectiveness of 122 126
 technic 123 ff
 oil injection causing eleoma 168
 for rectal procidentia 282
 Sedatives suppository form 57
 Sexual function in colostomy patient 208
 Shigellosis 232 f
 Sigmoid *see also* Colon
 adenocarcinoma of 195 ff
 diverticula of endoscopic diagnosis 291 f
 endoscopic examination of 31 ff
 lymphatic supply 23
 prolapse into rectum 284 ff
 treatment 287 f
 Sigmoidoscopy
 in cancer diagnosis 178
 in children 68
 in chronic ulcerative colitis 214
 in diagnosis of diverticula 291 f
 limitation and dangers 35
 technic 35 ff
 Situs *see also* Fistula
 pilonidal 295 ff
 Sitz bath technic 62
 Skin pettinal
 care in pruritus ani 263 ff
 excessive after hemorrhoidectomy 136

- grafting for recurrent pilonidal disease 316
- Sphincter
- excision in anal fissure operation 112
 - preservation
 - in anorectoplasty 117
 - in cancer resections 198 ff
 - spasm of
 - in anal fissure 105 f
 - diathermy in treatment 65
- Stricture
- anal
 - congenital dilatation of 75
 - galvanism in treatment 61 f
 - anorectal
 - congenital 73
 - after hemorrhoidectomy 133 f
 - rectal in lymphogranuloma venereum treatment 245
- Sulfadiazine for bacillary dysentery 233
- Sulfonamides
- for lymphogranuloma venereum 245
 - in ointment base for wounds 57 58
 - in ulcerative colitis therapy 220
- Suppositories as vehicle for medications 57
- Surfacaine with penicillin ointment 57
- Surgery *see also* specific operations
- abdominoperineal resection for anal cancer 182
 - anorectal
 - incontinence resulting from 289
 - postoperative diet 59
 - for cancer of colon 196 ff
 - minor anesthesia in 41
- Syphilis anal 247 ff
- chancre resembling fissure 109
 - diagnosis 248
 - differential diagnosis 249

T

- Tamponade for late posthemorrhoidectomy hemorrhage 146 f

- Tattooing of perianal skin for pruritus ani 263
- Teropretin for terminal cancer patients 205
- Thiersch wire splint procedure for rectal procidentia 282 f
- Thiouracil for ulcerative colitis 220
- Trauma
- anal incontinence caused by 289
 - coccygodynia caused by 272 f
 - with infection, in pilonidal disease 300
 - to lower bowel wounds from 324 ff
- Trichloroethylene for self induced inhalation analgesia 53
- Triple Sulfu Cream
- for hypertrophic proctitis 230
 - for postoperative wounds 58
- Trynazin ointment 57
- Tuberculids perianal 240
- Tuberculosis perianal 238 ff
- curability 240
 - diagnosis 240
 - treatment 241 f
- Tumors *see also* Cancer specific types
- benign 149 ff
 - classification 150
 - lymphoid (anorectal) 166 ff
 - carcinoid of colon and rectum 169 210
 - extrarectal 327 f

U

- Ulcer
- anal
 - in children 68
 - in fissure (chronic) 104 107 f
 - granuloma inguinale causing 249
 - with hemorrhoids operation for 137 ff
 - plastic operation for 109 ff
 - anorectal diet in 59
 - rectal (chronic) from irradiation 324
- Urea
- with sulfonamides in wound healing 58

